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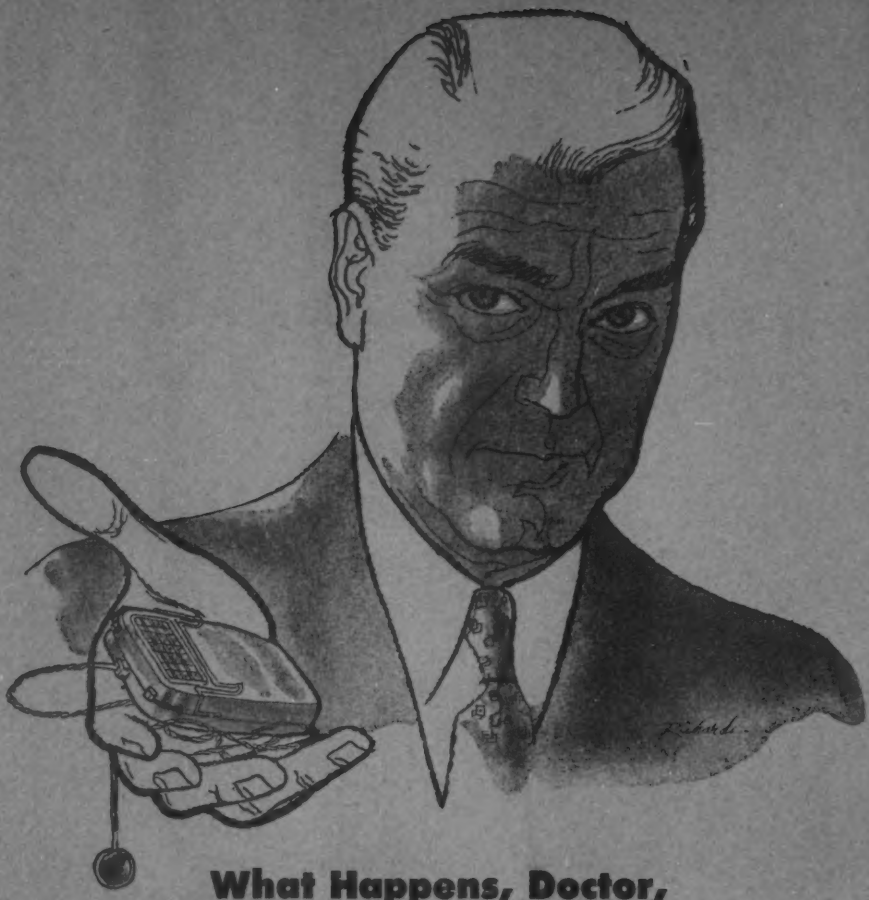

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TEN YEARS' EXPERIENCE IN THE USE OF TANTALUM IN FRONTAL SINUS SURGERY.*†

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The purpose of this paper is to report on cases which I presented at a meeting of the New England Otolaryngological Society in Boston, May 23, 1945,[1] and to describe some of the problems which have occurred in the use of tantalum foil as a method of maintaining drainage from the frontal sinuses after the Lynch operation.

In a paper published in 1942,[2] the author analyzed the causes for failure in frontal sinus surgery. Of 190 cases operated upon at the Massachusetts Eye and Ear Infirmary between 1931 and 1942, 123 were done by the Lynch technique; and of these 30.18 per cent required reoperation at a later date.

At the second operation the causes for failure were discovered to be the following: *1.* obstruction of the naso-frontal passage by adhesions; *2.* remnants of frontal sinus floor which later gave rise to undrained, infected pockets in the frontal sinus; *3.* incomplete ethmoidectomy, especially failure to remove orbital extensions of the ethmoid.

It was apparent that the wide opening provided by the Lynch operation from the frontal sinus into the nose did not

* Read at a meeting of the Eastern Section of the American Laryngological, Rhinological, and Otological Society, Inc., January 8, 1954, New York, N. Y.

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remain patent because of a tendency for the orbital contents to push the orbital periosteum toward the midline, where it came in contact with the medial wall. If this area had been traumatized it was inevitable that granulation tissue should form, and as all granulation tissue eventually is transformed into scar tissue it was obvious that this passage would become obliterated.

The technique which the author adopted was suggested by consideration of this surgical-pathological situation. If only the two surfaces could be kept apart by an inert substance, the granulation tissue of one surface would not have a chance to attach itself to the opposite surface, and the passage would then remain patent although narrowed in some cases. In searching for such a material, the metal tantalum was chosen because it proved to be inert in animal tissue and could easily be cut and fitted to the area to be covered at the time of operation. This was based on the work of Pudenz,[3] who had used tantalum plates in cranial defects in animals. He concluded that tantalum is a satisfactory material for repair of cranial defects as it has the desirable qualities of being non-corrosive, is inert in tissue, is non-absorbable, with an absence of toxic ingredients, and malleability.

The material for the cases reported in 1945 was a tantalum foil 1.5 cm. wide, 4 cm. long, and .00025 inches in thickness; sutured to the orbital periosteum by .003 inch tantalum wire.

During the past 10 years a total of 20 such cases have been performed, either by the author or under his supervision. This would seem to be a very small series, but it is to be noted that this technique is recommended only for such cases where it is definitely unlikely that the naso-frontal passage will remain patent after a Lynch operation. It is not in any sense a routine procedure for all cases. It should also be noted that during the past 10 years we have had the antibiotic drugs to control sinus infection. It is my belief that this accounts for a considerable decrease in the number of cases which we have had at Massachusetts Eye and Ear Infirmary during this period. I herewith present a follow-up on the cases which were presented in the paper in 1945.

Case 1. "A man, 42 years of age, entered the Nose and Throat Service at Massachusetts Eye and Ear Infirmary in September, 1943. Preoperative X-ray films showed a mucocele of the right frontal sinus with marked sclerosis about both frontal sinuses and a perforation through the anterior wall of the right frontal sinus. On October 23, 1943, a radical frontoethmoidectomy was done, and the lining of the frontal sinus together with the mucocele was removed. As the X-ray showed, there was a small perforation of the anterior wall near the supraorbital ridge. A strip of tantalum foil 1.5 cm. wide and 4 cm. long and .00025 inch in thickness was sutured to the orbital periosteum at about the position of the pulley

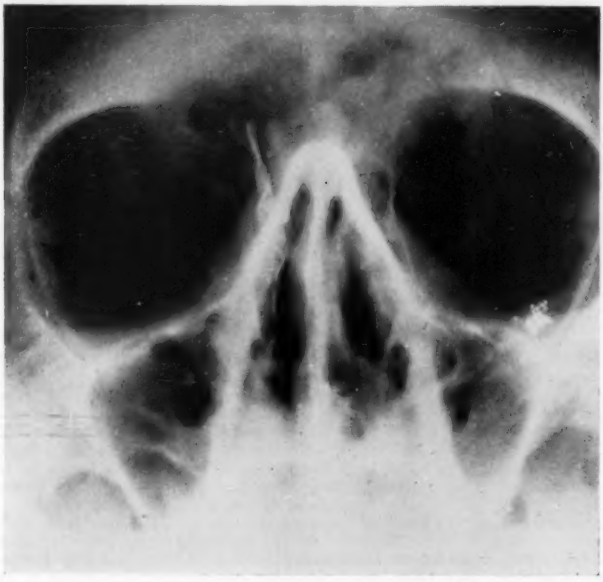

Fig. 1. Roentgenogram showing tantalum foil in place. This foil is attached at the upper end by tantalum wire to the orbital periosteum. The irregular outline is due to the fact that the sheet of tantalum is seen edge on. The foil extends downward through the area formerly occupied by the anterior ethmoid cells into the upper part of the nasal fossa.

(see Fig. 1). The foil was then carefully placed so that it was in contact with the orbital periosteum and extended medially and downward into the upper part of the nasal fossa. The suture material was .003 tantalum wire. The patient has tolerated this metal very well. There have been a few attacks of acute rhinitis, but the naso-frontal passage has remained patent and there has been no recurrence of the mucocele."

This patient reported to me again, at my request, in October, 1953. I had not heard from him for five years and naturally wondered whether he had had further difficulty. I was

pleasantly surprised when he gave a history of having had no trouble whatsoever with his frontal sinus since the operation. The tantalum was still in place; his nose was clean with no secretion. The only symptom he mentioned was that when he had occasional head colds his eyelids became slightly reddened, but this would last for only two or three days.

Case 2. "This was a girl, aged 15, who had had multiple left frontal sinus operations dating from March, 1942. Following each operation I attempted to keep the naso-frontal passage open by dilating the duct with a naso-frontal probe. In spite of repeated attempts at regular intervals, the passage narrowed until it had completely disappeared. On March 14, 1945, I reoperated and found that there was complete obstruction to drainage due to scar tissue and regeneration of bone near the nasal spine. The obstructing tissue was removed and tantalum foil was employed as in Case 1."

This patient has been under my observation since the first operation; she has had no more difficulty with her frontal sinus and the passage is open. Her only nasal difficulty since the operation was due to a nasal obstruction in the opposite nostril, which I corrected by a sub-mucous resection. She has had no recurrent infection in the frontal sinus.

Case 3. "This case illustrates the same difficulty. This was a patient who was referred to me for revisions of a right frontal sinus Lynch operation. The patient had been operated on for a fistula in an infected right antrum and for right chronic-fronto-ethmoiditis. The antrum had cleared up very satisfactorily, but the frontal sinus became blocked off by scar tissue. On May 11, 1945, I reopened the right frontal sinus through a Lynch incision. The adhesions were extremely dense and sprang from the region of the frontal spine to the orbital periosteum. The ethmoid was found to be completely exenterated by a previous operation. The sphenoid was entered easily and appeared to be in good condition. There was a little more bone to be taken off the ascending process. The frontal sinus contained mucopus and markedly thickened, infected mucous membrane. The floor had been well removed previously. I excised a great deal of organized scar tissue from the orbital periosteum and with a curette smoothed off some rough bone near the frontal spine. Above and below the frontal spine there seemed to be normal mucous membrane, which I hope will grow across the bare bone. Tantalum foil was used in this case with the same technique. The patient has had no reaction to the foil, and the frontal sinus has remained open."

About two years later the foil was removed, as the frontal sinus was open and the patient was symptom free. At last report he had had no further difficulty with his sinus.

Case 4. "This patient was a man, aged 64, who had suffered from right pansinusitis for many years. During the past winter, (1944), his nostrils had become completely blocked with nasal polypi, and had developed a persistent right frontal headache. In this case a right fronto-ethmoidectomy was performed. A slightly heavier tantalum foil, .0005 inch thick,

was used (see Fig. 2). His right antrum was not operated upon at this time. It has remained quiescent since the operation. He has a very clear, wide-open naso-frontal passage through which a probe passes easily into the frontal sinus."

This patient has been under my observation and was last seen at Massachusetts Eye and Ear Infirmary in October, 1953. An X-ray taken postoperatively is worth showing as it illustrates the position of the tantalum foil.

Fig. 2. The tantalum can be seen approximately in the position of the floor of the right frontal sinus. In this case one suture was placed near the upper end of the sheet of foil, and another suture at a point about 1 cm. from the lower end which lies in the upper part of the nasal fossa.

In all these cases the tantalum foil was tolerated without discomfort; in fact, even when touching the foil with a probe the patient experienced no pain or discomfort and actually seemed to be unaware of its presence in the nose. The naso-frontal passages have remained clear in every case where the tantalum foil has remained in the proper position; that is to say, between the orbital periosteum and the midline in the region of the frontal spine.

In all, there have been 20 cases in which I have had a chance to evaluate the results. I would like to present some of the defects and errors in this technique: *1.* error in position of the tantalum foil; *2.* removal of the tantalum foil under a mistaken idea that it is a foreign body and, therefore, may block the frontal sinus; *3.* displacement of the foil by injudicious probing so that it no longer remains at the proper level in the nose.

To illustrate the first error there are two cases: One, (P. R.), was operated upon July 13, 1945. This patient had pansinusitis of severe, chronic, hyperplastic type in which tantalum was sutured into both frontal sinuses. On the left side the tantalum remained in place satisfactorily; on the right the anterior edge of the tantalum slipped into the line of incision so that it prevented proper union of the skin edges. A fistula formed in this area which necessitated the removal of the tantalum foil (Feb. 15, 1946); however, in this case the tantalum had remained in place for seven months. By that time epithelialization had occurred, which prevented adhesions from forming.

The second was a private case in which I assisted another surgeon. Here again the foil slipped forward into the incision and had to be removed. In other words, the very properties of tantalum foil which recommend it for use in the naso-frontal area interfere with the proper closure of the incision. For this reason, I recommend placing the tantalum foil well within the frontal sinus, and if necessary closing the wound in two layers in such a way that the deep suture lies external to the anterior edge of the foil. In this way it will prevent the slipping of the material. It is also possible as in Case 4, to suture the foil at the upper and lower end, thus anchoring it securely to the periosteum.

To illustrate the second problem, that of removal of the foil on the theory that it is a foreign body, I describe the following case: This was an 18-year-old boy, (T. K.), who had had repeated frontal sinus infection eventually leading to a chronic empyema of the sinuses. One month after the operation he picked up an acute nasal infection. At that time he was living in a distant city, and the surgeon in attendance

removed the tantalum foil. The frontal sinus immediately became blocked with granulation tissue, so he presented me with the problem of further radical surgery. It seemed to me that in his case, if he again became reinfected, the whole story might be repeated; so I decided to obliterate his sinus completely and have a plastic repair of the defect done at a later date. On June 8, 1947, I obliterated the frontal sinus, and nine months later a plastic operation was performed by Dr. Edgar M. Holmes with a very satisfactory cosmetic result.

To illustrate the third problem, that of the foil becoming displaced by post-operative instrumentation, I quote the following case: This woman, age 51, entered Massachusetts Eye and Ear Infirmary with a diagnosis of right pansinusitis. Lynch operations were performed on October 30, 1950, and again on April 3, 1951, and as there was a strong likelihood that adhesions would form in the naso-frontal area, tantalum was sutured in the usual manner to the orbital periosteum. She returned again in November, 1951; at that time X-ray showed that the tantalum foil had been pushed back into the frontal sinus. A probe could not be passed into the sinus, as it was blocked by dense scar tissue. This patient was operated upon November 13, 1951, at which time an obliterative procedure was undertaken. The tantalum foil was removed and the patient had no further trouble in her sinus; however, the post-operative defect was a psychological hazard, so that on December 23, 1953, a bone graft was inserted to restore the contours of the forehead.

This brings up the question of the procedure which should be followed in difficult cases where re-infection is likely. As I have said before, the use of tantalum foil is not to be regarded as a routine procedure. If, in the opinion of the surgeon, an obliterative procedure such as I have just described offers a better outlook for the patient, it should be done; but where there is still a chance that the Lynch operation will succeed, and where the problem is one of maintaining naso-frontal drainage, I have found that tantalum foil has been most satisfactory.

I might mention three cases in which I was disappointed, not because of the tantalum foil, but because of other circumstances which occurred:

Case 1 was operated upon at Massachusetts Eye and Ear Infirmary by another member of the staff and seemed to be a fairly typical case for the use of tantalum. The surgeon removed a portion of the middle turbinate and sent it for pathological examination in a routine manner. He was not suspicious of malignancy which, however, proved to be the case. When the pathology report became available it was quite obvious that more surgery was indicated than originally contemplated; and, therefore, the tantalum was removed and further surgical procedures were undertaken.

Case 2 was a man with an alcoholic history. The surgeon who operated upon him was aware of this and thought that the pain which the patient experienced was of sinus origin, due to a pan-sinusitis. The Lynch operation was performed, and because of adhesions which made it difficult to maintain adequate drainage, tantalum was used. The pains continued, which later appeared to be due to alcoholic neuritis rather than to sinusitis.

Case 3 was a man who had a profound depression. In his case, although there was a severe sinus infection which was entirely cleared up by surgery, the depression became more marked. It was the opinion of the neuro-surgical service that the only relief for this patient was intra-cranial surgery, but the patient did not consent to this.

None of these three unfortunate patients had any complication from the use of tantalum.

SUMMARY.

In conclusion I again stress the fact that the use of any metal such as tantalum in frontal sinus surgery should be considered, not as a routine procedure, but rather in selected cases where it is difficult to maintain drainage from the sinus into the nose because of the tendency of scar tissue to form in this region. In my experience, when the tantalum has remained in the proper position, this has been successful. As in all surgery the proper selection of cases for any surgical procedure and the post-operative follow-up care is of great importance. I believe that in these cases, where it has been possible to keep track of the patients over a period of years, this method has proved successful.

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CONTACT ULCERS OF THE LARYNX.*†

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The clinical and pathological entity of contact ulcer of the larynx has been known to the laryngologist for many years. Jackson,[1] who gave the name to the condition in 1928, collected 127 cases over a period of 40 years. This would indicate his familiarity with this disease as far back as 1888.

This affection of the larynx is not common. In a period of 16 years, the writer has seen only 19 patients with nonspecific contact ulcers of the larynx. A study of this group has been made and is presented below. Particular importance is given to the value of vocal re-education as described by Peacher and Holinger,[2] for the treatment of these lesions.

By definition, contact ulcer is the superficial ulceration which may occur when one vocal process of any arytenoid cartilage comes in contact with the other vocal process. The ulceration may be unilateral or bilateral. The cause of the ulceration is trauma.

The traumatic factors have been described in detail by Jackson and Jackson,[3] and Jackson.[4] They have indicated that the trauma is brought about by a hammer and anvil type of mechanism; that is, the impact of the cartilaginous vocal processes against each other. The constant hammering is the cause of the ulceration.

Vocal abuse unquestionably adds a traumatic factor to the vocal process of the arytenoids. In certain individuals, who misuse their voices, the tips of these processes may bear a real strain on phonation. The muco-perichondrial covering of the tips is anatomically very thin, and it can be denuded when

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traumatized with consequent ulceration. This is a satisfactory explanation for the development of this nonspecific type of ulceration called a contact ulcer.

There are other causes of ulceration of the tips of the vocal processes. One of the sequelae of endotracheal anesthesia may be ulceration with granuloma formation in this location. The latter has the typical appearance of contact ulcer of the larynx; however, the etiological factor, as well as the treatment and prognosis, are different. The laryngeal lesions which may occur after endotracheal anesthesia offer no real diagnostic or therapeutic problem. Vocal rest, or vocal rest plus removal of the granulomas which may develop, usually effects a cure.

This is not the case with nonspecific contact ulcers. These have a different etiology and, therefore, the treatment and prognosis are different.

Johansen and Kaier,[5] made a thorough study of 21 patients with contact ulcers of the larynx. An autopsy was performed on 16 patients. There were 17 patients with pulmonary tuberculosis, in 14 of whom the ulceration was proved to be tuberculous, and in the remaining three, it was dubious. Typical nonspecific contact ulcers were demonstrated in four patients who had no pulmonary tuberculosis.

The work of Johansen and Kaier shows the importance of ruling out tuberculosis as a cause of contact ulcers.

The other important lesion which must be kept in mind is carcinoma. In my group of 19 patients, there was one case of proven squamous cell carcinoma. The patient had a superficial whitish ulcer over the tip of the right vocal process. A biopsy was taken and the diagnosis of carcinoma was established. X-ray therapy was given and the patient has remained well with an excellent voice for six years.

The pathological findings[6] in contact ulcer is that of a chronic nonspecific inflammation. The tissue examined may consist of new granulation tissue or fibrous inflammatory tissue, with or without cartilage in the bed of the ulcer.

SYMPTOMATOLOGY.

Contact ulcers usually develop in a patient who is not aware of any local or general illness. He has a sense of well being except for local symptoms to be discussed below. Occasionally, a history of a severe cold with paroxysmal coughing may be obtained. Also a patient may refer to overuse of his voice as a factor in the disease.

The most constant symptom is that of a sharp sticking pain in the region of the superior cornu of the thyroid cartilage on one side. The pain is aggravated by swallowing, and may be referred to the ear on the same side as the ulcer. It is very annoying and may be accompanied by a slight degree of hoarseness, cough and clearing of the throat. The patient is usually apprehensive because of the widespread publicity that has been given to the symptoms of cancer of the throat. As a result, he is apt to present himself for examination early in the course of the disease.

DIAGNOSIS.

The diagnosis of nonspecific contact ulcer of the larynx is not difficult to establish. Local symptoms are present and the patient will usually seek the advice of a physician, who refers him to the laryngologist. Occasionally, due to a lack of knowledge on the part of the examining physician, the diagnosis may be overlooked. As many as five different specialists examined one of the patients in this series and two specialists did not make the proper diagnosis.

By means of mirror laryngoscopy, typical lesions at the tip of one or both vocal processes are easily recognized. Ulcerations with or without granuloma are characteristic. It must be kept in mind that other specific lesions can produce ulceration or smooth-appearing projections in the posterior part of the larynx. Tuberculosis and carcinoma have especially to be kept in mind. The sequelae of endotracheal anesthesia must be ruled out. All patients with ulceration in this region should have an X-ray study of the chest and a biopsy before the diagnosis is established.

TREATMENT.

Before the work of Peacher and Holinger[2] in 1947, there seemed to be no definite plan of therapy. The role of vocal

abuse as a cause of these lesions was known, but no one had investigated the importance of vocal re-education in the treatment of the condition. Patients were placed on long periods of absolute vocal rest. Laryngeal examination, if accompanied by a biopsy, frequently aggravated the condition. Cauterization or excision of the ulcers followed by vocal rest left much to be desired.

After a regime of absolute vocal rest, the ulcers would usually heal, only to be followed by a prompt recurrence when phonation was permitted.

Vocal re-education has been a definite step forward. After the diagnosis has been established, speech therapy is begun in the presence of the ulceration. For the patient this is most important economically, as he does not lose any time from work because of vocal rest. For the laryngologist, the opportunity to observe his patient recover from a very annoying nonspecific disease is gratifying. There is much less tendency for the lesion to recur if vocal re-education is successful.

CLINICAL OBSERVATION.

There were 19 patients observed, all of whom were males. The youngest was 25 and the oldest was 57. Occupation did not appear to be important.

Before 1947, 12 patients were seen, and the treatment after the diagnosis was established consisted of a regime of absolute vocal rest and occasional local excision of granulation tissue and gentle cauterization of the base of the ulcer. In only one instance was an attempt made to remove the tip of the vocal process. This was followed by almost disastrous results, and the case will be reported below. In this group of 12 patients who were treated in this manner, as far as can be determined, most of the patients did well. As a rule none was worse and all showed a good response to vocal rest. The effect of local therapy to the lesion was doubtful.

Many of the patients became disturbed because of the long time which was necessary to accomplish a good result. These sought help elsewhere, and not without the blessing of the doctor, because he had nothing more active to offer them.

Since 1947, seven patients have been seen and given the benefit of speech therapy. All have had a good result from vocal re-education. In three instances the patients were at the transition period and had been on vocal rest with or without local therapy to the lesion. After Peacher and Holinger's report, vocal re-education was started on these three patients with an excellent result.

It takes a well qualified speech teacher to give the type of therapy required in this laryngeal disorder; however, every qualified teacher will not necessarily secure a good result with every patient. There is a strong psychological factor in this type of therapy, and some patients did poorly with one teacher but responded well with another teacher; therefore, vocal re-education should not be condemned if it fails. A change in teachers may be all that is necessary in order to effect a cure. The average time required for a good result to become evident is about two months. There was one instance of recurrence among seven patients treated in this way, and it was brief.

With the advent of vocal re-education, the laryngologist and the patient with contact ulcer are both more tolerant of each other.

CASE REPORT.

W. M., white, male, age 43, was examined on September 27, 1946, and found to have an ulcer on the tip of the right vocal process. A direct laryngoscopy was performed and biopsy taken. A nonspecific chronic inflammation was reported. He was placed on absolute vocal rest, but a granuloma slowly formed in the area of ulceration. Three months later, in January, 1947, it was necessary to remove the granuloma from the region of the vocal process of the right arytenoid. He did not show any improvement and formed another granuloma in the same location. This was removed in April, 1947. He was again placed on vocal rest but failed to improve. In July, 1947, the tip of the right vocal process was excised along with the granuloma. In spite of vocal rest, a granuloma reformed and increased in size to such a degree that it was attached to the posterior two-thirds of the right vocal cord and caused some respiratory difficulty. It appeared that a tracheotomy might become necessary. It was evident that the granuloma was infected, and in desperation the patient was referred for X-ray therapy. He was given six X-ray treatments of 150 Roentgen units each making a total of 900 units over a period of two weeks. The granulation tissue promptly receded and decreased in size. At this time, Peacher and Holinger's report came out. It was given careful study and the patient was referred to a speech therapist. Within a period of three months, his larynx healed and he has remained well without a recurrence to date.

SUMMARY.

Nonspecific contact ulcers of the larynx are not common. The important cause of the lesions is trauma, due to vocal abuse.

Differential diagnosis is based on the patient's history, the findings by indirect and direct laryngoscopy, X-ray study of the chest, and biopsy.

Treatment of nonspecific contact ulcers of the larynx should always include vocal re-education.

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THE OTOLOGIST'S ROLE IN THE INDUSTRIAL HEARING CONSERVATION PROGRAM.*

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Otologists in increasing numbers are enlisting in the struggle to mitigate the hazards of industrial noise and to conserve the hearing of exposed workers. Much has been written and said about occupational hearing loss and the conservation programs used in industry. It is commonly agreed that such programs, to be successful, require the cooperation of management and workers, health and safety personnel, and that a "team approach" is indicated.

Not only does the present writer endorse this opinion, but he would also seek to promote such teamwork through a candid exposition of the contribution which interested ear physicians can and should make to the properly conducted hearing conservation program. His observations and recommendations are based upon personal experience with hearing loss claims brought before the Wisconsin Industrial Commission, and with hearing conservation programs now in progress in the Wisconsin area.

The otologist who has interested himself in the many facets of occupational hearing loss is well suited to play a key role in the industrial hearing conservation program. As a result of his special training in the anatomy and function of the hearing mechanism, and his practical experience in the diagnosis and treatment of various hearing defects, he has acquired the skills needed for the proper management of a sound and purposeful program. He can offer realistic advice on such aspects of the industrial noise hazard as the following:

1. *On the "tailoring" of a Practical Program of Hearing Conservation to Meet the Needs of a Particular Plant.* It should never be overlooked that plants vary in their size,

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operation, production rates and acoustical characteristics, as well as in the number of workers. This means that each plant will have a "noise problem" which differs from its neighbor's or from plants which produce the same type of goods; therefore, the factors mentioned above, as well as others, such as the frequencies present in shop noises should be carefully considered. The program should neither be too costly nor require the worker to absent himself too long from productive work. Both workers and management must be instructed as to the need for and the purposes of the various procedures undertaken, if their cooperation is to be gained.

2. *On the Selection of a Suitable Room for the Conducting of Hearing Tests.* A room should be selected which is not too far from working areas, but which has, or can be treated to produce an ambient noise level low enough to permit the performance of practical hearing tests. A room with an overall noise level below 50 decibels is preferred but is not always obtainable in the industrial setting.

3. *On the Selection of the Audiometer and the Checking of its Performance.* In almost every plant a simple, but properly calibrated, and maintained, pure tone audiometer will meet all the needs for testing the hearing of workers in the plant. The accurate measurement of speech reception and discrimination tests, as well as the determination of bone conducting thresholds, requires optimum testing conditions and should be performed in the otologist's test rooms.

4. *On the Instruction and Guidance of Plant Personnel Conducting the Tests.* Experience has shown that the two most important requisites for the individual who is to perform the hearing tests are: *skill* and *interest.* Skill can be acquired through supervised practice, and interest can be stimulated by full instruction in the "whys and wherefores" of hearing and its measurement in the industrial setting.

5. *On the Evaluation of Pre-employment and Periodic Hearing Tests.* The interpretation of audiograms is a function best left to the medical man, as the one best prepared by training and experience to synthesize the medical and industrial histories with the results of hearing tests and otological examinations.

6. *On the Placement of Workers with Impaired Hearing.* This function is of particular importance where workers reveal significant or profound hearing losses at the time of hiring. It is essential that such workers be carefully examined and placed, after thorough hearing studies. Careful diagnostic differentiation between conductive, perceptive and mixed types of hearing losses also are of crucial importance in placing workers. The predominantly conductive deafness acts as "built-in ear plugs," while the predominantly perceptively deaf require careful watching less they be irreversibly aggravated.

7. *On the Selection, Fitting and Use of Ear Protective Devices.* Considering that any protective device to be inserted into the ear canal is essentially a foreign body, it becomes obvious that the supervision of this aspect of the hearing conservation program should be in the hands of medical personnel. It is imperative, too, that all necessary hygienic measures be taken.

8. *On the Susceptibility of Individual Workers to Occupation Hearing Loss.* Recently proposed test procedures suggest the feasibility of determining the susceptibility of individual workers to "noise deafness." There are differences, however, between the several proposed methods as to their reliability and time requirements.

9. *On the Detection of Willful Exaggeration of Either Hearing Ability, or Hearing Loss, by Workers.* Occasionally cases are encountered where a worker with appreciable loss, whose hearing has been tested several times in the past, will use his familiarity with the tests to "pass" a perfunctory screening test, and mislead his prospective employer as to his ability to hear. At other times workers will, for one of several possible reasons, respond in such a way as to exaggerate the extent and degree of his hearing loss. Either case can be detected and handled on an individual basis.

In addition to the above, the otologist participates in the medicolegal aspects of hearing claims as they arise. Some of the many thorny questions upon which he has been asked to render opinions are discussed on the following pages:

At what level of sound intensity, and over what period of exposure, does hearing damage take place? Until the subcommittees of the American Acoustical Society, and the American Academy of Ophthalmology and Otolaryngology, publish their final recommendations, only tentative figures are available for guidance. In general, it can be stated that the best damage risk estimates tend to be made in terms of the frequency spectra of factory noises, as well as the intensity. They show that decibel for decibel, high frequency sounds are more damaging and that sounds that contain many frequency components are less damaging than sounds which liberate the same energy within a narrow band of frequencies.

What was the status of the claiming employee's hearing when he came to work for the employer? This question was of crucial importance when the first wave of hearing loss claims appeared. At present there is no valid reason why employers should not require routine audiometric examinations as part of the pre-employment physical examinations. When cases arise, employers should, as a matter of defense, be able to establish degree and extent of any hearing loss present, when the worker was hired, the hearing status at the time of claiming, and the cause of any decrement in hearing between these times. The need of otological advice in such cases should be evident to all concerned.

What tests should be used for assessing the hearing loss? It is pretty generally agreed that older methods such as watch ticks, coin clicks, and whispered and conversational voice are too subjective to be of any value. Calibrated pure tone audiometry is much to be preferred to any of the above, but it is sometimes objected to, on the grounds that it measures acuity for tones rather than for useful everyday speech. Calibrated speech audiometry in the hands of skilled practitioners, is the method of choice, and is rapidly gaining endorsements from authoritative professional groups.

What formula should be used to evaluate the hearing test results in terms of percentage of hearing disability? The American Medical Association formula of 1947 has, in the past, been used by many otologists for evaluating pure tone audiograms; however, valid objections to the use of this for-

mula have arisen, especially in connection with hearing loss claims based upon decreased acuity for high frequency tones. It is argued that such claims can be liberally awarded when this formula is used, although the claimant has lost little of his ability to follow normal conversation. In New York State, the Fletcher Point Eight (.8) method has been used. There is every indication, however, that for medico-legal purposes, speech audiometry will soon supplant pure tone audiometry with the sanction of national authoritative professional societies. It is expected, however, that pure tone audiometry will be used as an adjunct to calibrated speech audiometry.

Which of several hearing test results obtained by several doctors on different days should be used to evaluate the hearing loss? This question gains importance in cases involving claims for alleged occupational hearing loss. As the claimant is examined several times by different doctors in rooms of varying acoustical characteristics, and becomes familiar with the test procedures, variations in test results are commonly found. This gives rise to a diversity of opinions as to which of several different hearing curves should be accepted as most valid and reliable.

The Wisconsin Industrial Commission has ruled that when two or more differing audiometric results are found within a short period of time—performed under similar testing conditions—the test showing the best hearing acuity shall be used.

Are some workers more susceptible to noise induced hearing loss than others? That some workers are more susceptible to damage and loss than others can no longer be doubted.

How is the permanence of the hearing loss to be determined? The problem of permanency of hearing loss is difficult to solve. It is well-known that part of the total amount of measureable hearing loss that comes from noise exposure is recoverable. If the employee is removed from the noisy environment some improvement in hearing can be expected. The American Academy of Ophthalmology and Otolaryngology has expressed the following opinion regarding the question of permanency: "Hearing loss, caused by prolonged exposure to loud noise, may be considered as permanent six

months following complete removal of the individual from the area of loud noise." A more definitive answer may be expected after an analysis of observations now being made by several researchers.

What consideration should be given to the age factor? Loss of hearing, which is an accompaniment of advancing age, has been given the name of presbycusis. This hearing loss in older people is supposedly due to simple atrophy and degeneration of the end organ. The hearing loss is of the perceptive type, affecting the higher frequencies and rarely involving the lower scale. This high tone loss has been demonstrated to increase with each decade of life, and appropriate allowances can be made for the amount of hearing loss which can be expected, due to normal aging of the hearing mechanism. An allowance of 5 per cent is made for individuals over 50 years of age and 10 per cent for the 60 and over age group.

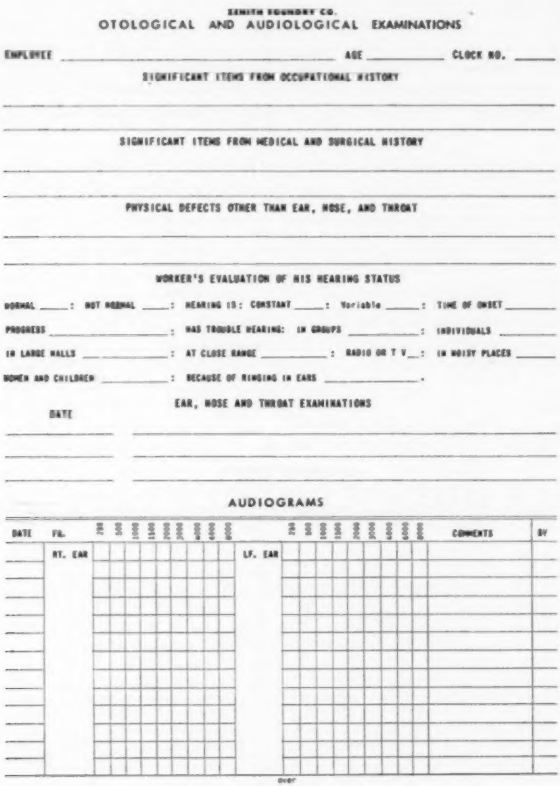
The hearing conservation program begins with a thorough study of the noise levels found in the various working areas. A preliminary survey reveals those areas where sound levels reach intensities that are potentially damaging. Then a more detailed analysis of these areas is made to determine the intensity, duration and frequency characteristics of the overall sound pattern.

Such an analysis indicates the amounts of energy present in the more traumatizing frequency bands, and determines the amount and type of acoustical treatment and individual worker-worn ear protective devices. Where noises are predominantly of the impact type, it is often necessary to use the cathode ray oscilloscope to obtain an accurate measurement.

The next step is to obtain a convenient and accessible room suitable for the performance of hearing tests. When a sufficiently quiet room cannot be found, it is sometimes possible to treat an existing space with acoustical materials and obtain an acceptable room for practical hearing tests.

The existing medical records are studied, and additional forms are prepared, which will allow the serial recording of periodic otological and hearing tests, and pertinent items of medical and industrial history for each worker (see Figs. 1

through 3). Experience has shown that charts such as these which allow the serial recording of initial test results, together with the findings of a dozen or more "follow-up" audiograms on a single sheet, are less bulky and less subject to loss



ZENITH FOUNDRY CO.

OTOLOGICAL AND AUDIOLOGICAL EXAMINATIONS

EMPLOYEE _____ AGE _____ CLOCK NO. _____

SIGNIFICANT ITEMS FROM OCCUPATIONAL HISTORY

SIGNIFICANT ITEMS FROM MEDICAL AND SURGICAL HISTORY

PHYSICAL DEFECTS OTHER THAN EAR, NOSE, AND THROAT

WORKER'S EVALUATION OF HIS HEARING STATUS

NORMAL ____: NOT NORMAL ____: HEARING IS: CONSTANT ____: Variable ____: TIME OF ONSET _____

PROGRESS _____: HAS TROUBLE HEARING: IN GROUPS _____: INDIVIDUALS _____

IN LARGE HALLS _____: AT CLOSE RANGE _____: RADIO OR T V __: IN NOISY PLACES _____

WOMEN AND CHILDREN _____: BECAUSE OF RINGING IN EARS _____.

EAR, NOSE AND THROAT EXAMINATIONS

DATE

AUDIOGRAMS

DATE | FU. | 250 500 1000 2000 3000 4000 6000 | COMMENTS | BY

RT. EAR | LT. EAR

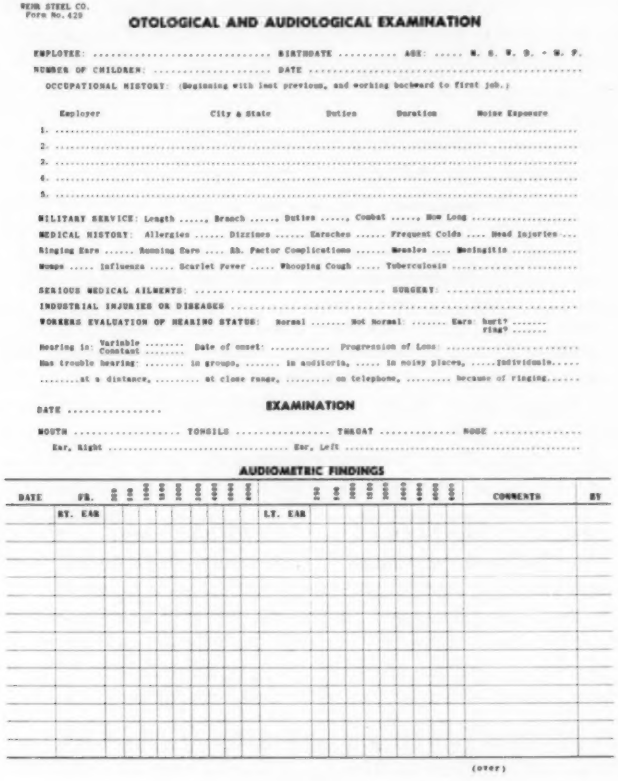
over

Fig. 1.

than a group of single index cards for each test on the same individual. Since the results of all hearing tests given a worker are grouped together on a single sheet, any changes in hearing status are quickly and easily noted.

After a short and intensive course of instruction from the

otologist, followed by supervised practice, plant personnel are ready to perform audiometric tests on workers. Close supervision is required at first, and is gradually relaxed as the tester develops skill and judgment.



WEHR STEEL CO.
Form No. 429

OTOLOGICAL AND AUDIOLOGICAL EXAMINATION

EMPLOYEE: ..... BIRTHDATE ..... AGE: ..... M. S. W. D. - W. P.

NUMBER OF CHILDREN: ..... DATE .....

OCCUPATIONAL HISTORY: (Beginning with last previous, and working backward to first job.)

	Employer	City & State	Duties	Duration	Noise Exposure
1.					
2.					
3.					
4.					
5.					

MILITARY SERVICE: Length ....., Branch ..... Duties ....., Combat ....., How Long .....

MEDICAL HISTORY: Allergies ..... Dizziness ..... Earaches ..... Frequent Colds .... Head Injuries ....

Ringing Ears ..... Running Ears .... Rh. Factor Complications ..... Measles .... Meningitis .....

Mumps ..... Influenza ..... Scarlet Fever ..... Whooping Cough ..... Tuberculosis .....

SERIOUS MEDICAL AILMENTS: ..... SURGERY: .....

INDUSTRIAL INJURIES OR DISEASES .....

WORKERS EVALUATION OF HEARING STATUS: Normal ..... Not Normal: ..... Ears: hurt? ..... ring? .....

Hearing is: Variable ..... Constant ..... Date of onset: ..... Progression of Loss: .....

Has trouble hearing: ..... in groups, ... in auditoria, ... in noisy places, ...Individuals.....

.....at a distance, ... at close range, ... on telephone, ... because of ringing.....

DATE .....

EXAMINATION

MOUTH ..... TONSILS ..... THROAT ..... NOSE .....

Ear, Right ..... Ear, Left .....

AUDIOMETRIC FINDINGS

DATE	RT. EAR 250	500	1000	1500	2000	3000	4000	6000	8000	LT. EAR 250	500	1000	1500	2000	3000	4000	6000	8000	COMMENTS	BY

(over)

Fig. 2.

The results of all hearing tests are reviewed and evaluated by the otologist, and all workers showing significant deviations from normal hearing are listed. When enough workers with abnormal hearing are listed to justify it, the otologist arranges to spend part of a day examining them in the plant. It has been found that this practice, in other words. the oto-

logist's visiting the plant, is more economical and convenient than sending the workers to his office. In typical small plants the otologist need hold examinations only two or three times a year.

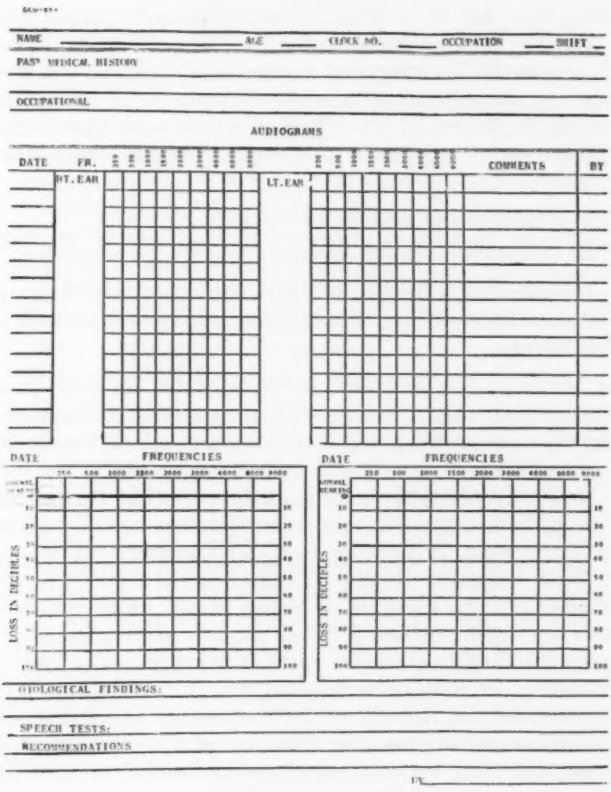


Fig. 3.

The value of his supervision and guidance is amply demonstrated by the type of errors which he routinely detects. He must determine whether hearing loss is due to acoustic trauma or to normal aging of the ear, and whether a worker's

hearing loss is real or only apparent. He is familiar with the effects of ambient noise, active respiratory diseases, obstructions to the ear canals, etc.

He quickly determines whether the worker is consciously or unconsciously exaggerating, either his hearing ability or his hearing loss, and he is alert to detect "cross-hearing," which can lead the inexperienced tester to credit a totally deaf ear with serviceable hearing; but in the final analysis, the worth of the aurist's contribution to the hearing conservation program can be judged by the extent to which he helps to achieve the three major objectives of a second hearing conservation program:

1. to conserve the hearing of workers,

2. to avoid unnecessary economic loss to the employer,

3. to collect the facts and data which will enlarge our knowledge of the defects of industrial noise, and how they can be overcome.

At all times, the otologist should be aware, as much as are his co-workers, that this is a joint effort requiring the earnest cooperation of workers and management and of the medical and allied professions. It will be his responsibility to interpret the data in hearing records made as part of the program and to share his findings with his colleagues. At the present time, the results of hearing tests on thousands of workers have been tabulated in the Wisconsin area alone. Together with similar data collected in other states, these statistical studies are beginning to fill in the gaps in our knowledge and to make speculation about the facts of hearing loss in industry unnecessary. It is to be hoped that more otologists will interest themselves and become active in attempts to overcome the effects of industrial noise. On the basis of my own experience, I would definitely recommend those interested to take part in our efforts. There are both challenges and opportunities aplenty in this field, and time alone will tell whether we as otologists have been equal to them.

208 East Wisconsin Avenue.

THE ROLES OF SENSATION LEVEL AND OF SOUND PRESSURE IN PRODUCING REVERSIBLE AUDITORY FATIGUE.

J. DONALD HARRIS, Ph.D.,
New London, Conn.

The determination of unusual susceptibility to auditory fatigue has long been proposed as a prophylactic step in a noise control program. One of the first workers to standardize a test for individual differences in susceptibility was Smith,[3] who studied threshold shifts at 4000 cps. caused by stimulation at 2000 cps. for two minutes, finding fatigue to range from 10 to 56 db. This wide range opened the possibility of an industrial program designed to reject those who suffer the greatest fatigue. Peyser[2] determined threshold at 256 cps., stimulated at 80 db intensity for five minutes, and 30 seconds later again determined threshold. Of 80 subjects, seven showed threshold shifts of more than 5 db.

Wilson[4,5,6] has energetically attacked this problem with rather large numbers of subjects. He early discarded testing at 256 cps., finally standardizing a stimulus of 2048 cps. at 80 db intensity for five and eight minutes. Threshold shifts at 4096 cps. were determined as an indication of fatigue.

By comparing irreversible audiometric loss caused on the rifle range with amount of fatigue on his tests, Wilson claimed to have established a good predictive relationship between the two functions. If true, Wilson's test, or a similar one, could well serve in industry and in the armed forces to reduce the number of auditory casualties and compensable cases.

A question arises in a test of this sort whether it is well to place the stimulating frequency at a certain intensity for all subjects, or to place it a certain number of decibels above the individual's own threshold. It is clear that if a subject has a threshold 10 db better than "normal," and another subject has a threshold of 10 db less than "normal," the same

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physical intensity of, say, 80 db on the audiometer scale will furnish sensation levels of 90 and 70 to the two subjects respectively; now, if the fatiguing effect of a tone depends not on its physical intensity but on its perceived *loudness*, then the effective stimulus conditions for the two subjects will have been greatly different, and we shall find we have not given comparable fatiguing conditions as we wish to do. This study proposed to investigate the relative effectiveness of loudness and intensity in producing auditory fatigue. This done, the data should help to standardize a fatigue test to attack the problem of predicting irreversible trauma.

PROCEDURE AND APPARATUS.

Thresholds on a total of 66 subjects were obtained at 2048 and 4096 cps. with a Sonotone audiometer calibrated in this Laboratory. The range of hearing loss at 2048 cps. was — 10 to + 65 db. A fatiguing stimulus at 2048 cps. was led to each subject for five minutes from a Hewlett-Packard oscillator connected to the Sonotone earphone. Sound pressure level (as measured in the National Bureau of Standards 9A Coupler) was maintained at 97 db (this is the equivalent of 80 db above "normal" on American audiometers at this frequency). The stimulus was terminated by a two-way switch that immediately fed a 4096 cps. tone to the earphone from the Sonotone audiometer. The level of this tone had previously been set 20 db above the individual subject's threshold for that frequency. The 4096 cps. tone was turned on and off in ¾-second intervals; the pulsing nature of this tone made it easier to distinguish in the presence of high-tone tinnitus, which very often follows stimulation of the sort used here. As soon as the 20 db sensation level tone was heard, the subject tapped a microphone of a wire recorder, whereupon the experimenter reduced the tone 5 db and waited for another tap; and so on until the time taken to return to within 20, 15, 10, 5, 2.5, and 0 db at threshold had been recorded on the wire. In this way, a complete picture of every man's recovery curve could subsequently be drawn.

If a subject heard all tones as soon as presented, we assumed that his recovery curve was complete in less than 10 seconds, the time it required to move the audiometer dial from

20 to 0 db in the steps as given above. In that case, one or more repetitions of the whole procedure were given, in which the dial was set initially at 10 rather than at 20 db.

RESULTS AND DISCUSSION.

Effect of Sound Pressure Level Versus Sensation Level.

Since all of our subjects received the same acoustic stimulus, distributions of various indexes of fatigue will serve to specify the effect of sound pressure level. Table I gives the distributions of times to return to own threshold, and to within 5 and 10 db of own threshold. From these data one

TABLE I.

INDIVIDUAL DIFFERENCES IN RECOVERY TIME AT 4096 CPS. TO STIMULATION WITH 2048 CPS. AT 97 DB SOUND PRESSURE LEVEL FOR 5 MINUTES.

TIME TO RECOVER TO:					
1. Own Threshold		2. Within 5 db.		3. Within 10 db.	
Time	N	Time	N	Time	N
0-2′ (1′59.9″)	28	0-30″ (29.9″)	29	0-20″ (19.9″)	39
2′ - 4′	7	30″ - 60″	6	20″ - 40″	4
4′ - 6′	4	60″ - 90″	2	40″ - 60″	0
6′ - 8′	4	90″ - 120″	2	60″ - 80″	1
8′ - 10′	2	120″ - 150″	2	80″ - 100″	2
10′ - 12′	1	150″ - 180″	1	100″ - 120″	1
12′ - 14′	0	180″ - 210″	2	120″ - 140″	0
14′ - 16′	0	210″ - 240″	2	140″ - 160″	0
16′ - 18′	2	240″ - 270″	2	160″ - 180″	0
				180″ - 200″	1

can fairly well infer the effect of sound pressure level *per se* on the recovery curve. Note that about 58 per cent of our first 48 subjects completely recovered in less than two minutes. If, however, we consider only those 25 men whose hearing at 2048 was *better* than "normal," we find that only 40 per cent of this exceptionally acute group recovered in less than two minutes. Evidently the data of Table I are strongly influenced by selection of subjects, and we conclude that sound pressure level is no sure predictor of fatigue effects.

When we consider the relative effect of sensation level, it is necessary only to group our subjects according to individual threshold at 2048 cps. It will be recalled that the threshold

range of our subjects at this frequency was minus 10 to plus 65 db ; therefore, the sensation level of the 80 db tone on the audiometer actually ranged from 90 db to 15 db SL. One would assume that the subject who is presented with a 15 db tone will show little fatigue in comparison with the subject who hears a 90 db tone. Fig. 1 shows mean curves for 36 of our subjects divided into five groups according to their pre-stimulation threshold (the remaining 12 of the 48 subjects who took this test had hearing losses at 2048 cps. which did not fit into the categories of Fig. 1).

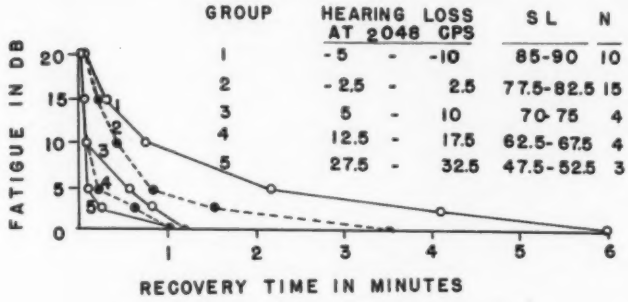

Fig. 1. Effect of Sensation Level on Recovery.

One can see the very significant differences among at least the first three groups with highest sensation level. Complete recovery time for the highest sensation level group (85-90 SL) is 360 seconds; for the next highest group it is 212 seconds and for the third highest group it is 80 seconds. The other three groups whose hearing is within 10 decibels of normal at this frequency, all show such rapid and similar recovery curves that one can say, when the tone is about 60 db sensation level and below that there is little difference in recovery from this stimulation, as measured by our technique, and that there is no significant amount of fatigue. At 60 db SL and less, the difference occasioned by sensation level can not be assessed by this technique, but above this level sensation level must be taken into consideration before predicting any sort of susceptibility to fatigue based on a stimulation set at 80 db above "normal" on the audiometer.

Individual Differences.

The usual wide range of individual differences appears in our data. The raw data for six representative subjects in Group 2 of Fig. 1 (those who were "normal" ± 2.5 db) are shown in Fig. 2. The recovery time to own threshold ranges from seven seconds to 16 minutes. Note that this wide variability occurs even among subjects who were given substantially equal stimulation, whether measured by sound pressure level or by sensation level.

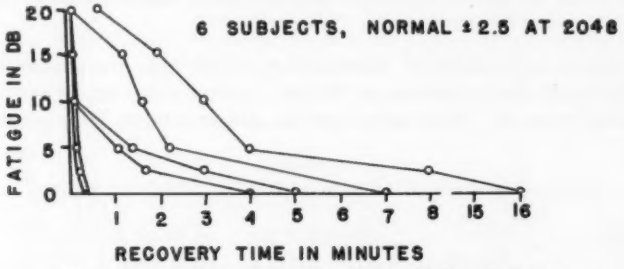

Fig. 2. Individual Differences in Susceptibility to Fatigue.

The effect of type of deafness was not investigated in this study, though we did include a few partially-deaf individuals to show with especial clarity the difference between equal sensation level and equal physical intensity. The problem of type of deafness has, however, been well covered by Huizing,[1] who gave the same sensation level of stimulation to patients with about the same hearing loss (50-60 db) but with quite different types of disorder. Pure conduction deafness yielded 7-12 db fatigue (about the same as normal-hearing individuals on the identical test), and inner ear deafness without recruitment yielded 5-11 db , while inner ear deafness *with* recruitment yielded 19-22 db. Huizing concludes that the fatiguing effect of a stimulus depends among other things upon its *loudness.*

Comparison of Wilson's and the Present Study.

Wilson used subjects with no hearing complaints and his threshold loss was no more than 30 db at 4096 cps. in either ear. His experimental procedure was to determine the subject's threshold at 4096 cps. in both ears, then to expose the

ears (one at a time) to 2048 cps. for five minutes at "80 db intensity." Threshold was "immediately" taken at 4096 cps., and again ½, 1, 2, 3, 5, and 8 minutes following the start of recovery, or until 10 db from threshold was reached. (A deviation of less than 10 db from the pre-stimulation threshold was considered to be within experimental error.) This procedure was then repeated on the opposite ear.

Wilson arbitrarily divided his subjects into three groups depending on the time required to return to within less than 10 db of threshold (we presume this to be subject's threshold, not necessarily audiometer "normal").

When, regardless of threshold at 2048 cps., we group our data in the same fashion as Wilson, comparable results appear as in Table II. This table reveals disagreement between the

TABLE II.
COMPARISON OF WILSON'S AND THE PRESENT STUDY.

Group	Percentage of Cases Wilson	Present Study	Returned to within less than 10 db of Threshold in:
1	69.2%	97.9%	Less than 2 minutes
2	14.8	0	2-3 minutes
3	16.0	2.1	3 or more minutes

two studies. Only one of our cases did *not* meet Wilson's criterion of no fatigability (i.e., return to within less than 10 db of own threshold in less than two minutes), whereas, 30.8 per cent of Wilson's cases did not meet this criterion (sum of Groups 2 and 3).

These discrepancies can hardly be due to differences in the 2048 cps. threshold between the two experiments. It might be thought for example, that most of Wilson's subjects were better than "normal" on the audiometer, and then in consequence if 80 db over "normal" were used, the actual sensation level would be well over 80 db; however, we used ten subjects who were better than "normal" by at least 5 db, and who consequently received stimulation at 85 db or more sensation level. None of these required more than two minutes to reach 10 db over their own threshold. We may further consider our 15 subjects who were "normal" $\pm$ 2.5 db, and so received a stimulus at 80 db sensation level (within experimental error), and compare this group with Wilson's

on the assumption that what he did was to stimulate at 80 db above each subject's threshold. Only one of this group (7 per cent) did not meet Wilson's criterion, again as compared with his 30.8 per cent. The differences then are not those concerned with variations in sensation level or sound pressure level of stimulation.

Wilson's percentages may be expected to be somewhat larger than ours, since he included a man in the group dictated by the more susceptible ear if there was a difference in the ears. We used only one ear from each subject.

The writer feels, however, that most of the difference in the studies is probably due to the tinnitus which easily can partly obscure a continuous tone. It will be recalled that ours was a pulsing tone which serves to avoid penalizing those not-rare

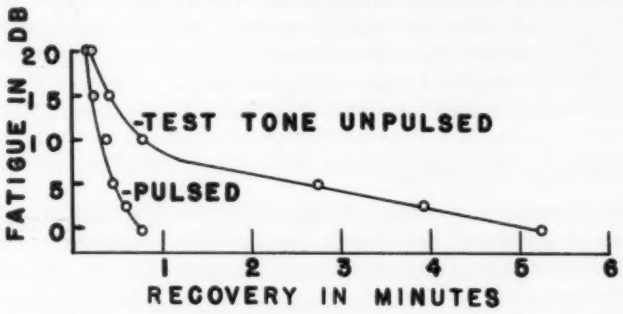

Fig. 3. Effect of Pulsing Test Tone on Recovery Time.

individuals with tinnitus at or near 4096 cps. after loud stimulation. Wilson mentions a number of subjects, in whom tinnitus suddenly ceased after 2-3 minutes, whereupon their thresholds returned sharply to normal.

In order to investigate this point, we used an entirely new group of 18 subjects, and gave them stimulation at 2048 cps. at 80 db sensation level for five minutes, immediately following with a 4096 cps. test tone, either pulsed as described previously, or continuous. A direct comparison in this group of 36 ears can then be made in the recovery time as investigated by a pulsed versus a non-pulsed tone. The data is summarized in Figure 3, and show that the median recovery time is

of the order of 45 seconds for a pulsed tone, but is of the order of 315 seconds for a continuous tone. The differences between the two techniques are somewhat greater if the arithmetic mean is taken, rather than the median. This for the reason that several of the subjects had a tinnitus at the 4000 cycle region, which interfered for many minutes with hearing the unpulsed tone at all. These extreme subjects, however, do not unduly influence the data in the median measure of central tendency.

Another way to consider the data of these 18 subjects is to compute the percentage of those who returned to normal, according to Wilson's criterion. With the pulsed technique, all of the 36 ears had returned to within less than 10 db of threshold in less than two minutes. On the other hand, with the unpulsed technique only 22, or about 61 per cent, returned to this criterion. The other 39 per cent required longer time. It may be observed that this percentage is approximately what Wilson found, as revealed in Table II.

Criterion of Fatigability.

In choosing a criterion of fatigability for the practical purpose of screening individuals particularly susceptible to fatigue, Wilson settled on the threshold shift of 10 db or more, after two minutes of recovery. We feel that a somewhat more stringent criterion would perhaps be preferable. As the raw data show, a one-minute interval would have picked out five ears, or about 10 per cent, as being especially susceptible. This percentage is in general more usable in a personnel selection program than the 2 per cent picked out at the two-minute interval (see Table II).

An alternative is to record the time to return to within 5 db. of own threshold. Table I shows that only about four to five minutes would be required for even the most susceptible. Choosing this level would not, then, prolong the test unduly; and the distribution of recovery time is wide enough to serve in a statistically controlled fatigability-research program.

We conclude that either criterion could serve, the threshold after one minute, or the time to return within five db of threshold, depending upon the convenience of available apparatus.

SUMMARY AND CONCLUSIONS.

A test for screening fatigue susceptible individuals was investigated. A tone of 2048 cps. was sounded for five minutes at 97 db sound pressure level (80 db on the audiometer). The individual's recovery curve was carefully plotted at 4096 cps., the region most affected by such stimulation. Results were analyzed in terms of sensation level (data of their own threshold), and physical intensity. Sensation level proved the more important parameter.

Considerably more rapid recovery was found than reported previously by Wilson, who used, as a criterion of fatigue, inability to hear a tone 10 db over own threshold after two minutes recovery. The present study shows a better criterion to be, either the time in seconds to return to within 5 db of own threshold, or the residual hearing loss after one minute.

It is of considerable importance to use a pulsing tone rather than a steady tone in determining threshold during recovery from stimulation, in order to minimize the irrelevant masking effects of the high tone tinnitus which very often follows stimulations such as used here.

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CHEMICAL BURN OF THE ESOPHAGUS. REPORT OF A CASE TREATED WITH CORTISONE AND PENICILLIN.*

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The majority of chemical burns of the esophagus fall into the class of second-degree burns. Stumboff,[1] in a series of 603 cases, found that burns of the second degree constituted 63 per cent of the total.

In a second-degree burn, necrosis of the mucous membrane and muscular layer takes place. There is a sloughing of the necrotic tissue followed by the formation of granulations. Collagen fibers appear and form the basis for scar tissue formation. These fibers appear usually about the second week following the burn. Unless the formation of scar tissue can be prevented or controlled, a stricture of the esophagus results.

Stothers,[2] in 1952, reviewed the prophylaxis of stricture formation following burns of the esophagus, and stated that it was generally conceded that early bouginage by the Salzer method is the best means available to prevent stricture following chemical burns. Dilation by any means usually takes months and possibly years.

Krey[3] by animal experiments, showed the importance of secondary infection in the formation of cicatricial strictures. Inflammatory changes produce granulation tissue, which in turn is transformed into scar tissue. Limitation of inflammatory reaction by resting the burned esophagus until epithelization is complete, retards the formation of scar tissue. In a group of animals having a burn of the esophagus and treated by gastrostomy, sulfonamides, penicillin and streptomycin, epithelization took place in 35 days, and only moderate stenosis was observed. Stenosis of the esophagus sufficient to

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prevent feeding developed in three other control groups. The first group received no treatment; the second group received antibiotics; the third group received antibiotics and an indwelling tube.

Rosenberg, Kunderman, Vroman and Moolten[4] administered large doses of cortisone to rabbits with chemical burns of the esophagus and found that fibrous stricture formation was remarkably diminished; however, many of the animals died as a result of infection. Infections were rare in the control group.

In a later series of experiments, Rosenberg and associates[5] found that the administration of penicillin with cortisone to rabbits having chemical burns of the esophagus prevented stricture formation, and also improved the mortality figures. In a small number of rabbits receiving penicillin and cortisone, spontaneous rupture of the esophagus occurred in the second or third week, usually resulting in mediastinitis and death.

Weisskopf[6] treated a child with a severe lye burn of the esophagus. He employed cortisone, terramycin and a Levine tube. The esophagus healed without stricture formation or other complication. He then produced lye burns of the esophagus in dogs. The treated animals received cortisone and terramycin. The controls received no medication. Animals in the control group showed marked granulations of the esophagus in the early stage, and developed strictures if allowed to live over five weeks. No stricture developed in the treated animals.

Recently we had under our care a patient with a severe second-degree burn of the esophagus due to sulfuric acid. The results of treatment were so satisfactory as to merit a report.

REPORT OF A CASE.

R. D., white, male, age 25, was admitted to the Neuropsychiatric Service on July 14, 1953, because of a depressive state, in which he had attempted suicide by drinking sulfuric acid used for storage batteries. This episode had occurred on the day prior to admission. He had received emergency treatment at a local hospital and was then transferred to the V. A. Hospital. Upon admission temperature was 100.6°, pulse

80, and respirations 20. The next day the temperature was elevated to 101°. Blood pressure was 126/80. He complained of sore throat and inability to swallow. Liquids when taken by mouth were vomited almost immediately.

Because of the dysphagia, he was seen in the E. N. T. Clinic on July 15. Examination of the pharynx showed the mucosa to be markedly congested. In the hypopharynx the mucosa was edematous as well, and there was profuse saliva stagnant in the valleculae. The patient was started on procaine penicillin 300,000 units daily by intramuscular injection, and cortisone 50 mg. twice daily. He was given bland liquids by mouth. Sore throat and dysphagia were less marked in 48 hours.

On July 20, 1953, esophagoscopy was performed. This was seven days after the acid had been ingested. The mucosa of the entire esophagus was congested and edematous. At a level of 27 cms. from the upper teeth there was marked sloughing of the mucosa. Near the hiatus the mucosa was again found to be sloughing in the entire circumference of its lumen. The sloughing areas bled on passage of the esophagoscope.

A polyethylene Levine-type feeding tube was passed through the esophagoscope into the stomach and the esophagoscope withdrawn. The feeding tube was then brought out through the left nostril by means of a catheter. The patient was fed a liquid diet high in protein and meeting the caloric, mineral, vitamin and fluid requirements. The cortisone and penicillin were continued daily. There were no physical complaints. On August 6, the feeding tube was removed and the cortisone and penicillin discontinued. By this time the patient had gained 15 pounds in weight.

Esophagoscopy on August 18, showed a normal mucosa with no evidence of scarring, nor of stricture. This was five weeks after the patient had swallowed the acid.

The patient was discharged from the hospital on August 19, 1953, with instructions to return for periodic follow-up; however, all efforts to induce him to return for esophagoscopy and esophagram were unavailing. He was interviewed at his home in December, 1953, by a social service worker. At that time he stated he was having no difficulty with his regular diet, and that his weight had remained constant. This was five months after the trauma to the esophagus.

COMMENT.

Acid burns of the esophagus are less frequently the cause of stricture of the esophagus than are lye burns; however, from the appearance of the esophagus in this case, we feel that in the ordinary course of events, stricture would have resulted, or months and possibly years of dilation would have been required to prevent it.

Polyethylene tubing is chemically inert and causes no tissue reaction. It maintains the patency of the lumen, puts the ulcerated mucosa at rest and allows the patient to take his normal fluid and caloric requirements without discomfort. Broad spectrum antibiotics may be given with the tube feedings. Its use probably also lessens the chance of perforation.

Weisskopf[6] used terramycin in his case and also in his experimental animals to control secondary infection; we used penicillin. Apparently, either broad or narrow spectrum antibiotics are efficacious in controlling secondary infection and resulting granulations. Used alone, they probably will not effectively prevent stricture formation.

Cortisone lessens the formation of collagen fibers and resulting scarring; and when used alone, in animal experiments, resulted in high mortality. In our case it was administered along with the antibiotic from the third day after the burn until the twenty-fourth day. The dosage used was small, because we did not wish to submit the patient to the numerous and daily laboratory tests required by hospital regulations when a full dosage of cortisone or ACTH is administered.

SUMMARY.

A case of second-degree burn of the esophagus, which was treated by cortisone, penicillin and polyethylene feeding tube, and which healed without stricture formation is reported.

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SUBACUTE MASTOIDITIS.

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Ballance[1] quoted Celsus as having said, "Though we should not refuse to give modern authors due credit for their discoveries or happy imitations, it is none the less just to restore to the ancients what properly belongs to them."

This brief paper will deal with the clinical, audiometric and radiologic considerations for surgical intervention in the recent and recurrent mastoid infection. Clearer concepts on this subject are needed. The old indications for simple mastoidectomy in the pre-antibiotic era are obsolete. Every patient with a chronic ear wishes something had been done surgically during the simpler phases of the infection. There is no more shock to a simple mastoidectomy than to a tonsillo-adenoidectomy. Every purulent, acute otitis media is accompanied by mastoiditis, and the indications for surgery and the anatomy of the mastoid have become confused by the administration of antibiotics. The candidates for modified and radical procedures are younger each year.

"Colds," sore throats and pink eardrums constitute many of the indications for penicillin and other antibiotic administration. The early treatment of these symptoms, by whatever specialty, is most important for the patient.

There is a general tendency to minimize the solid clinical judgment of the otolaryngologist in evaluating the need for surgery.

The final evaluation of the antibiotics and their promiscuous administration awaits the passage of time. The agreeable sophistry is advanced that the patient gets well faster with antibiotics, but has he acquired any natural immunity during their administration?

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BACTERIOLOGY.

In an address before the staff of one of our hospitals on this same subject in August, 1951, attention was called to the dangers of moniliasis from antibiotic administration. That danger undoubtedly still exists; if not a factor in otology, witness the rumbling of millions of feet of intestine.

An editorial[2] in a reputable medical publication, stated: "The replacement of respiratory or gastro-intestinal flora with antibiotic resistant strains of pathologic organisms such as hemolytic *staph. aureus* and hemolytic streptococci is potentially a much more serious problem than overgrowth of normally saprophytic fungi or yeastlike organisms."

Not only are routine ear cultures bringing these strains to dock but there are increasing numbers of *proteus mirabilis, proteus vulgaris, ps. aeruginosa, alcaligenes faecalis, B subtilis e. Coli* and *aerobacter aerogenes* infections of the middle ear and mastoid.

Ellard Yow[3] called attention to the increased importance of *Proteus* and *Pseudomonas* as pathologic organisms, isolating these from normally sterile body fluids during antibiotic therapy. He felt that surgical drainage and removal of non-vital tissue were important in controlling these infections.

An increasing number of well-known authors have been pleading for surgical treatment of surgical lesions, *i.e.* pus-filled cavities, but their cries go unheeded.

A case was recently seen of purulent mastoiditis, of ten years' duration, with an intact drum except for a sieve-like group of perforations high posteriorly, which revealed *proteus mirabilis.* The patient had been given everything including cortisone. The aural discharge may still persist. Another patient in whom a bilateral secondary mastoidectomy revealed a middle ear and antrum on both sides—behind intact drums —filled with the thick and typically tenacious secretion suggestive of *ps. aeruginosa,* was reported "no growth." The bacteriology laboratory may have poor laboratory technique

or, more significantly, the cultural characteristics of bacteria are likewise changing.

CLINICAL PICTURE.

The case seen by the otolaryngologist usually has a history of recent antibiotic administration.

Chief among the presenting symptoms of this type of infection is *pain*. It may be located in the classical areas of old —tip, emissary vein, and/or root of zygoma. On specular examination it may be seen surrounding the bony canal in the absence of external ear infection, and manual pressure may elicit a wince or exclamation.

The *psychodynamic* factor must be excluded, as this idea clouds the symptom as frequently as do the antibiotics. Whether inflation relieves the pain is a differential point.

The last "no growth" case mentioned above was believed to be of psychosomatic origin, although surgery (in spite of intact normal appearing drums), revealed cortical erosion of an unoperated side, bilateral, healed peri-sinus abscesses, thick discharge in aditus, middle ear and antrum, and intact trabeculae of bone. Operation resulted in complete relief of all symptoms, notably night pain.

Night pain often awakens the patient from sound sleep—as might a night terror symptom. It may be of a "thumping" character, if a sterile discharge is imprisoned in the tip from escape to aditus and is being replenished by secretion from seemingly "normal" mucoperiosteum of cell.

Temperature is no longer a prominent finding unless the chronic ear has progressed to jugular sepsis, brain abscess, subperiosteal abscess, or Bezold's type.

Aural discharge is not the reliable surgical indicator it once was. There is still a tendency to believe that this is part of a "tubal catarrh," from the perforation location. Many feel that every aural discharge is accompanied by mastoid disease. It is important to note its chronicity as it continually recurs, despite seemingly free intervals, usually in the same ear.

Page[4] feels that a continued but scant discharge through a minute "pouting" perforation is an indication for drainage of the tympanum through the mastoid to preserve the hearing and to prevent a chronic purulent otitis media. The white count may or may not be significantly elevated.

An objective finding of some worth is the appearance of the drum. There is frequently lysis of the structure with a slight ring of tissue; again, there may be a *wet tissue paper* appearance with evidence of moisture. It is sad to note such changes in patients before their fifth birthday.

Rutherford[5] has given a clear, concise and forthright picture of the management of acute otitis media.

The combined picture is one of much indecision on the part of the physician, a confused patient, and recurrences of trouble in the same ear over long periods of time. In one case still under observation in Pennsylvania (mostly by hearsay) this picture has persisted without surgical intervention for thirteen years. The greatest unanswered question is why there should be disagreement—not with patient or parent—but among physicians, as to the *need* for surgery.

AUDIOMETRIC.

The audiologic findings in these patients is nearly always constant if the drums are intact. In most patients, seen at bi-annual visits, who finally submitted to surgery and who are no longer bothered with ear pains or psychosomatics, certain audiometric findings are prominent.

In two ears in the same patient which seem equally affected, *i.e.* with retracted, lustreless, red or scarred drums and with same clinical findings, the audiometer will serve to reveal the more involved ear. Pullen's[6] observations are of value.

We know that a patient with an acute otitis media will have a decreased air conduction with possibly no change in bone conduction, but as the mastoid becomes involved, his categories of altered bone conduction are important. Pullen lists the large cellular mastoid which signifies extensive loss of bone conduction and the small, underdeveloped and sclerotic

mastoids in which bone conduction is normal or only slightly impaired. In a chronic otitis media, "healing" through sclerosis, Pullen's observations will prevail; however, in the large, cellular mastoid filled with the "sterile" or "banked fire" of a mastoiditis, bone conduction will be better in the more extensively involved ear presumably because the insulating effect of the air cells is thus removed, and vibratory sound is transmitted more readily through the bone with added granulation or thick cell wall.

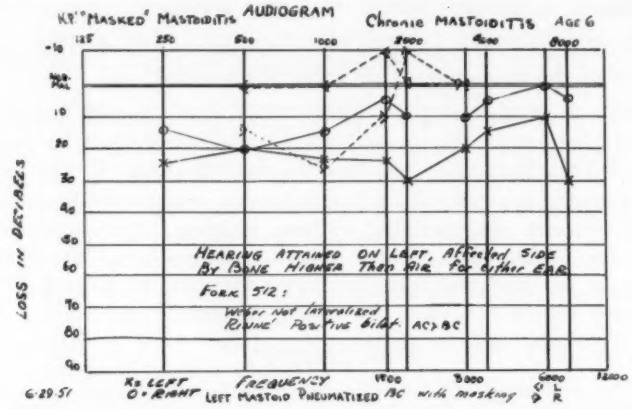

Fig. 1: K. P. Pre-operative audiogram on patient with diagnosis of left masked mastoiditis shows a very definite increase in Bone Conduction on left; diminished air conduction on left in absence of signs or symptoms. This increased bone conduction may be a characteristic audiometric feature of masked mastoiditis following long antibiotic therapy.

This observation has been confirmed at surgery in at least four patients and in many more when the radiologic comparison of mastoids was utilized to ascertain the ear of greater involvement on area of increased density basis.

Fig. 1 shows a typical observation in an operated patient. Hearing by bone conduction in the poorer ear was better than by air conduction in either ear.

RADIOLOGIC.

Boies and Younger[7] write of abnormal pneumatization of the temporal bone. It is their belief that in a population between the ages of five years and early adult life approximately

14 per cent probably have a middle ear mucosa in one or both ears that is not the thin mucoperiosteum of the well pneumatized mastoid. In a personal communication Boies states that he does not feel that lack of pneumatization, due to the effect of infection on a pneumatic structure, will necessarily carry a hereditary factor.

Two Spanish-American families have presented themselves, in which the mother and both sons have a discharging ear. No other members of these families have been seen with this complaint of aural discharge, yet here are two mothers and four sons, with three right ears and three left ears affected in a family pattern.

Boies feels that 14 per cent of such cases will show a deviation from normal. Since the postulation of Witmaack's theory there has been no unanimity of opinion on the significance of the sclerotic mastoid. Some prominent writers feel that it is an effort to heal a diseased area.

Tumarkin[8] quotes the theories of Diamant and Dahlberg regarding pneumatization and the pattern of the adult being stamped on the newborn; that acellular mastoids were simply normal variants genetically determined—a "sterilizing hypothesis" carried out with greatest care. Cawthorne, quoted in the same article, feels that chronic otitis media is the result and not the cause of an acellular mastoid.

Whatever the ultimate end, it is far better to dissociate theory from practical observation. The majority of patients will demonstrate a pneumatic mastoid by X-ray and, until proven otherwise, any deviation from this dawn-of-history-to-present pneumatic development should indicate mastoid pathology. Establishing time of onset of symptoms from history, physical findings, and the clinical picture, are still most reliable. X-ray studies are important for future evaluation.

Ersner, Alexander and Myers[9] decry the tendency to rely solely on X-ray in "irreversible mastoiditis" and Barrett[10] was among the first to point out the first reliable roentgenologic indicator in "masked" mastoiditis, of penicillin therapy.

In the young individual with an antrum at the two o'clock position there is an area of translucency surrounded by thickened sinus and dural plate. There is ordinarily an area of softening along the posterior canal wall with elevation and irritation of periosteum to produce clinical evidence of specular pain formerly accompanying "sagging of the canal."

For other than otologists this is frequently interpreted by the radiologist as "no evidence of cellular breakdown," when even acquainted with presence of an aural discharge in the radiographed patient. Law was the only radiologist who saw his temporal bone cases at surgery.

In the young adult with thick, hazy cellular partitions, some antral softening and increased density of bone surrounding the posterior canal, if the comparative film is normally thin in trabeculae with changes manifest equally in tip views, one may feel that this represents a pathologic condition. These are the insidious changes in the mastoid picture which, as stated by Ersner, are frequently overlooked. These are the mastoids which may terminate in mastoiditis or one of its intra-cranial complications.

Boies and Younger feel that abnormal pneumatization is an important factor in the chronicity of certain cases of sterile fluid in the middle ear; that it may play an important role in those children developing suppuration in the antibiotic era.

It is my feeling that the sterile fluid found in many of these mastoids at surgery is a pathologic sequence of antibiotic, sterile irritant fluid with bony change and progression to chronicity. How else may we account for the yellow color of the thin fluid entrapped in cells with apparently normal periosteum and filled with granulation? In most instances the radiologic haziness is revealed at operation as bleeding granulation, with thick, bony septae and pin-point areas of granulation. All of this is on the surface of the normally delineating boundaries of the mastoid cavity.

It must be remembered that even before antibiotics, the radiographic interpretation was not the sole criterion of operability and, like Tamari and Henner,[11] a uniform approach

to the need for surgery is advocated before the ear becomes chronic and to indicate "when surgery is indicated."

THE SURGICAL POSITION.

The incidence of acute otitis media terminating in acute mastoid surgery (simple mastoidectomy) in the pre-antibiotic era was approximately 2 per cent. There have been decreased numbers of mastoidectomies at institutions like the Manhattan Eye, Ear and Throat Hospital since 1947. There is a need for better surgical indications in the outlying areas where reliance is still being placed on the antibiotic alone, without preliminary myringotomy, when needed.

A tabulation of mastoidectomies done by this writer in the five-year period, August 8, 1948 to 1953, is listed for 5,500 new patient office registrations.

One case was done in 1948, three in 1949, ten in 1950, 19 in 1951, 12 in 1952, and 3 thus far in 1953. Three of these were radical mastoidectomies; the remaining cases were simple mastoidectomies. The incidence of those coming to surgery is less than 1 per cent and these were by no means, all acute cases. Only six were subperiosteal abscesses and one was a brain abscess. The downward trend for 1952-53 reflects an attitude of indifference and inability to "reform" those who rely on antibiotics.

Probably as many refused surgery as had the operation, and the incidence of those who needed surgery has apparently been reduced but very little from the pre-antibiotic era. Those refusing surgery rarely return for evaluation, and it is this group which surgery would benefit in arresting the progress of pathology. Those having surgery usually have tried everything else, and surgery comes at the stage of urgency—a rare thing in antibiotic days, or when all other courses of treatment have been exhausted. In most cases the hearing is down; drums are nearly gone and only complications are prevented.

There have been innumerable authors urging that a pus-filled lesion is a surgical lesion, and nonetheless true for the mastoid infection. It is comparable to osteomyelitis only in the fact of bony involvement, and is worse from a standpoint

of the tremendous vascularity of the bone, surrounding vital structures and the tragic loss of hearing in the young from damage to the delicate mechanism.

There may be only a remnant of drum left before mastoidectomy is suggested by the general physician, and the case is referred to the otolaryngologist. Simple precedures will no longer produce drum healing.

We have urged operation on all patients with subperiosteal abscesses, as well as those exhibiting marked bony change, without treatment by the antibiotics for a month or more, in which the conservative measures have failed to produce a dry ear.

We have had no serious complications save brain abscess to convince the patient as to the wisdom of surgery. We have also felt that in the patient with recurrent attacks of otitis media, in the same ear, surgery is the wiser course. When these attacks number two or more in a year or less, and despite previous conservative or antibiotic therapy, surgery is indicated. In one of my cases which has been seen at yearly intervals the pediatrician has averaged a course of penicillin monthly for each "pink" eardrum. There appears to be an increase of adhesive processes in middle ears of the younger patients so treated by repeated medical "abortions."

Surgery is an individual proposition. The listless child with vague ear symptoms and X-ray evidence of mastoid disease responds to surgery very well.

Resolution of the acute mastoiditis may occur quite naturally when the drum perforates, and constant drainage is established. There has been permanent damage.

Is there such a saving in health, time or money to the patient given repeated courses of antibiotics, as compared to a clean-up trip to the operating room? There is no saving to those who have had several bouts of antibiotics and long periods of malaise, vague abdominal discomfort, diarrheas, dementias and dermatitis. These are the patients who finally wonder if surgery is not the answer, and how disappointing it

must be for such a patient to have his otolaryngologist suggest further antibiotic therapy.

Modern surgery can compete safely with medicine alone in every aspect of mastoiditis. We may decry this reliance on antibiotics and the pernicious therapeutic philosophy outlined by Wesley Spink[12] that : (quoted verbatim)

1. Antibiotics are highly effective for many common infections.
2. That these are readily available, inexpensive, easy to administer and non-toxic.
3. With a febrile patient, cause unknown, give an antibiotic.

Ask any mother whose child had repeated bouts of middle ear infection, nosedrops, tonsillectomy, allergy treatments, radium treatments, X-ray treatments, *ad infinitum*, how much happier ultimate surgery has made her and her child.

The most important surgical justification in the operating room as to need for surgery is probably the color of the lateral sinus and whether or not it is lusterless and violaceous.

Only *anesthesia* need complicate surgery, and this should be open drip ether for children and Flagg administration for the adult, with change to the smaller can after thirty minutes, to prevent stagnation and rebreathing. With or without burrs, and regardless of route, the surgical procedure of simple mastoidectomy is of great value.

CONCLUSIONS.

It is doubtful whether the incidence of patients requiring or meriting careful consideration of the need for surgery has been materially reduced by antibiotic therapy.

Many children are needlessly becoming hearing problems.

The antibiotic, which has its justly earned place in the therapeutic armamentarium, should be used as an adjunct to rather than a substitute for surgery in lessening the dangers of operating through an infected field and preventing complications.

Only surgery can drain the tip or remove the dense central plate blocking the aditus.

Though symptomatology has been minimized by the administration of antibiotics, it is doubtful whether the true pathology has been greatly altered or reversed thereby.

In the patient with two or more recurrent bouts of otitis media in the same ear within a year or less, conservative procedures having been previously carried out, and with X-ray evidence of damage present, one should consider surgery.

Once mastoidectomy has been decided upon and procedure in progress is completed, the surgically treated mastoid should clear up quite satisfactorily without the administration of antibiotics. If these are administered, they should be discontinued as soon as possible, to observe the natural changes in temperature and white count.

We, as otolaryngologists should approach the serious and confusing problem boldly rather than abandon a tried and true surgical principle.

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TUBEROUS SCLEROSIS. A CASE REPORT.

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The purpose of this report is to add to the literature an example of a rare syndrome and to call the otologist's attention to points wherein he might be of help in the diagnosis.

Tuberous Sclerosis is characterized clinically by the triad of mental retardation, epileptic seizures, and a peculiar type of cutaneous lesion called sebaceous adenoma. It has been referred to as one of the congenital ectodermoses and is frequently associated with Von Recklinghausen's disease. Other congenital neoplastic formations, such as tumors of the kidney and retina, are frequently associated, since it is a blastomatous ectodermal defect.[1]

A comprehensive review of the literature is difficult because of the association of tumors of other origins; however, it is interesting to note that significance of the histological alterations was first appreciated in 1912, when Bundeschuh gave us complete studies based on the embryological concepts of Ranke. Different aspects of the triad are emphasized by different authors; however, our primary interest is centered on the cutaneous lesion. In 1891 Pringle[2] reported a case of congenital adenoma sebacium citing that the tumors growing on the face were small papillary growths varying in color from pale to dark red. They ranged in size up to one cm. and had a butterfly distribution over the nose and cheeks.

E. L. S., a 24-year-old white male, reported to this clinic complaining of intense itching, pain, and discharge of the ears. This proved to be a rather severe case of diffuse external otitis which responded quickly to treatment. The most notable findings were many small papillary tumors arranged in a butterfly facial distribution and filling the external canal. This observer's curiosity was not aroused sufficiently at that time to remove one of the tumors for biopsy. A few weeks later a local urologist[3] reported to us that he had removed a large hamartoma of the kidney and that the diagnosis of tuberous sclerosis was considered. The patient revealed that he had had Jacksonian type seizures intermittently since childhood. At this point, the pathology report was obtained on one of the ear tumors. It is recorded:

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"Microsection of the biopsy removed from the region of the right ear reveals moderate increase in the number of sebacous glands with increased keratinization in follicles in these glands. Frequently the follicles are dilated and contain desquamated, keratotic debris. The necks of the

Fig. 1.

Fig. 2.

sebaceous glands are lined with stratified squamous epithelium. About these glands there is a moderate chronic inflammation. It is much more marked just beneath the epidermis than about the glands. Even in this location it tends to be rather focal in distribution. There are no changes present that would suggest malignancy and as far as can be determined, the primary change is that compatible with sebaceous adenomas."

In summary, a brief review of the literature and an illustrative case of tuberous sclerosis is presented. It behooves the otolaryngologist to delve deeper into the history of patients presenting small fibromatous tumors of the external ear.

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POSTGRADUATE COURSE IN OTOLARYNGOLOGY

The Department of Postgraduate Medicine of the University of Michigan Medical School announces the Otolaryngology Conference to be given at the University Hospital, Ann Arbor, Michigan, April 15-17, under the direction of Dr. A. C. Furstenberg, Chairman of the Department of Otolaryngology, University of Michigan Medical School.

Guest Lecturers: Dr. J. A. Hilger, St. Paul, Minn.; Dr. H. P. Schenck, Philadelphia; Dr. Paul Holinger, Chicago; Dr. K. M. Day, Pittsburgh; and Dr. Lawrence R. Boies, Minneapolis, Minn.

Resident Lecturers: Dr. A. C. Furstenberg, Dr. J. H. Maxwell, Dr. J. E. Magielski, Dr. Thomas Francis, Dr. Jerome Conn, Dr. E. R. Harrell, Jr., Dr. R. B. Sweet, Dr. H. E. Sloan, Jr., and Dr. Irving Blatt.

For further information, address: Dr. H. H. Cummings, Chairman, Department of Postgraduate Medicine, University Hospital, Ann Arbor, Michigan.

FROZEN SECTION BIOPSY IN PERORAL ENDOSCOPY.

FLAVIO APRIGLIANO,† M.D., and ERNANI TORRES,* M.D.,
Rio de Janeiro, Brazil.

In the last years peroral endoscopy has become a routine procedure of examination. The specialist is able to make a very satisfactory and accurate examination of the larynx, trachea, bronchi and esophagus, but a large number of lesions located in these structures can not be actually diagnosed without the help of peroral endoscopy. Benign or malignant tumors, inflammatory lesions can be visualized, examined and a biopsy taken for the necessary histologic diagnosis. This undoubtedly is one of the greatest advantages of peroral endoscopy. If, due to circumstances, the biopsy in a few cases fails in the first attempt to bring out sufficient material for histologic diagnosis, it is necessary to repeat the endoscopic examination; and in a large number of cases this is not very well accepted by the patients.

To avoid such inconvenience at Casa de Saude Sao Miguel, we have been obtaining very good results during the endoscopic examinations by using routinely the frozen sections technique for histologic diagnosis of the biopsy material. The tissue obtained by the biopsy forceps is immediately taken to the pathologist, working in a laboratory close to the operating room, where it is sectioned, stained and examined under the microscope. The histologic diagnosis by this method is given to the endoscopist in a period of time varying from two to three minutes. If the biopsy is negative or insufficient, the endoscopist still has time and opportunity to take one or more pieces of tissue from the lesion without further inconvenience or risk to the patient.

† From the Department of Broncho-Esophagology of the Casa de Saúde Sao Miguel, and of the Hospital I.A.P.E.T.C.

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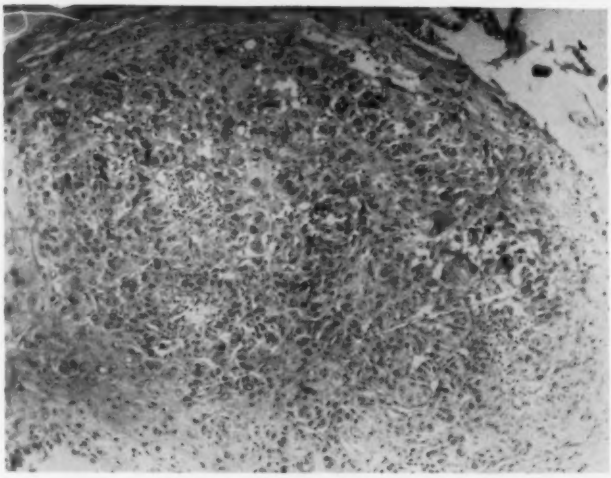

Fig. 1. Larynx.

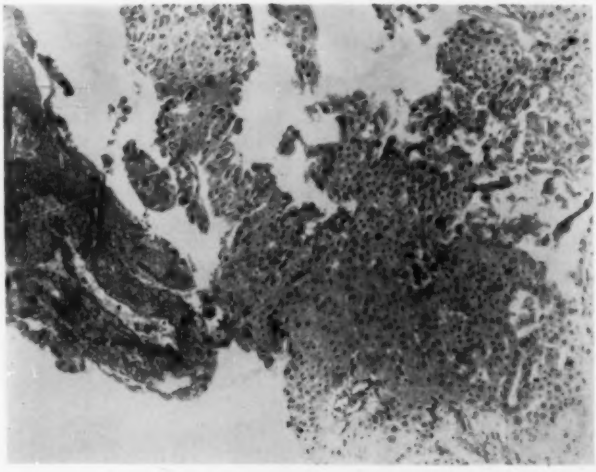

Fig. 2. Bronchus.

By this method, at the Casa de Saude Sao Miguel, from 1950, we had an opportunity to examine and diagnose cases of

lesions located in the larynx, trachea, bronchi, esophagus and base of the tongue.

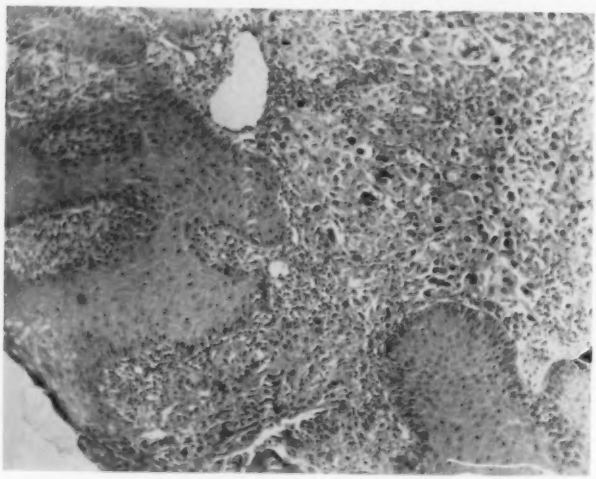

Fig. 3. Esophagus.

The histologic diagnosis and location of the lesions are shown in Table I.

In three cases of our series, two or more successive biopsies were necessary to reach a correct diagnosis. In these cases the delay of a few minutes caused no inconvenience to the patients. In 31 cases, the first piece of tissue taken by the biopsy forceps was sufficient for an immediate, correct histologic diagnosis. In three cases, two fragments were necessary, and in two cases, three fragments were necessary.

Table I.

Location	No. of Cases	Benign Tumors or Inflammatory Lesions	Malignant Tumors
Larynx	5	2	3
Bronchi	16	6	10
Esophagus	14	1	13
Base of Tongue	1	–	1
	36	9	27

TECHNIQUE.

The tissue obtained by biopsy forceps, is immediately taken to the pathologist. We use a frozen microtome to make the sections. We emphasize that with little practice small pieces of tissues can be very well sectioned in the frozen microtome, giving as good and as satisfactory sections for diagnostic purposes as large fragments. We prefer the Terry stain, or "neutralized polychrome methylene blue," which in our opinion has the following advantages:

1. Fixation is not necessary.
2. It is a vital stain and stains in few seconds.
3. It is extremely useful for diagnosis of malignant cells.

The sections so stained are covered by a coverslip and examined under the microscope. The same block of tissue is afterwards run through 10 per cent formalin, alcohol, xylol, etc., imbedded in paraffin, sectioned on the microtome, stained by routine hematoxylineosin, and the slides are kept in the laboratory files after being checked. Even the original sections can be stained afterwards by hematoxylineosin, after the necessary steps through 10 per cent formalin, alcohol, xylol, etc.

We consider the method, as described above, absolutely reliable. It is not necessary to bring out the advantages of this simple procedure to the endoscopist, surgeons and patients.

SUMMARY.

The authors advise the use of the frozen sections method for histological diagnosis during endoscopic examinations. They believe this method is absolutely reliable giving results identical to other methods, (paraffin sections), with the advantage of shortening the time necessary for histologic diagnosis.

In our cases, in which biopsy was possible, no patients left the operating room without diagnosis.

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Rio de Janeiro, Brazil.

PERCEPTIVE DEAFNESS TREATED WITH VITAMIN B-COMPLEX AND AMINO ACIDS— 100 CASES.

JOHN H. CHILDREY, M.D.,
San Jose, Cal.

Previously the failure of Vitamin B-Complex to influence the course of progressive perceptive deafness was reported.[1] Since then, in addition to the B-Complex, amino acids and histamine acid phosphate have been given over a 10-year period, and herewith is presented a report on 100 patients with perceptive deafness, tested before treatment was started and again an average of 9.79 months afterwards.

Since all these patients had the same type of deafness and were treated much the same, composite audiograms are the only accurate measures of the effect of therapy. While isolated cases reported improvement after treatment, either regarding tinnitus, which was a primary symptom, or the hearing loss, it seemed better to depend upon the audiograms which are precise and the only sure measure of curbing the indiscriminate use of unestablished forms of therapy.

Perceptive deafness is not an uncommon accompaniment of middle age, and occurs in various occupations in which there is prolonged exposure to intense noise. It may follow the use of drugs such as streptomycin or quinine, and is seen after high fevers or as a complication of meningitis. Since World War II its incidence is greater, due to the increase in the number of men and women working in heavy industry and serving in the armed forces, and the otologist now sees more young individuals with occupational deafness.

That noise is recognized as an occupational hazard, is indicated by cases on record in which damages were recovered by individuals deafened as a result of their employment. Industrial concerns and the armed services should obtain audiograms (other tests for hearing are not sufficiently exact) on

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all employees, and on those in service, at the time of hiring or the beginning of service, and again at the termination of employment or of service.

While deafness is recognized as a hazard in operation of motor vehicles in 36 States and the severely deafened might be forbidden to operate motor cars in those States, this restriction is probably too seldom enforced. It should be a national requirement that the hearing acuity of applicants for drivers' license be at least roughly tested with whispered or spoken voice before a license is granted.

Perceptive deafness is usually progressive, as is suggested by the composite audiograms on five untreated cases observed over an average period of two years, in which there was an increased hearing loss of 12.8 per cent binaural. Spontaneous recovery from perceptive deafness is remote, and "it is doubtful if any organ of the human body has as little power of recuperation as the organ of hearing."[2] In the 100 cases presented here there was increased hearing loss of 2.5 per cent binaurally, after 9 months, and while treatment did not result in improved hearing, it may have slowed the progress of the deafness; although probably not enough to justify the expense and effort expended.

Williams and associates,[3] by restricting the thiamin intake to an average of 0.35 mg. daily for 110 days were able to show development of polyneuropathy, consisting of defects of the sensory nervous pathways, loss of tendon reflexes and paralysis of the muscles of the legs, which after many weeks responded to large doses of thiamin. They concluded that the polyneuropathy was the result of late severe, rather than early mild, deficiency of thiamin. No mention of the organ of hearing is made. Kopetsky[4] was unable to help cases of deafness with thiamin alone, but advocates the use of the whole B-Complex, amino acids, trace metals, thyroid and pituitary extract, histamine desensitization and other measures.

The treatment used in the cases reported here consisted of large doses of amino acids and B-Complex orally, B-Complex parenterally, and histamine intravenously. When occasionally the amino acid product in liquid form was poorly tolerated, it was given in powder form. Most patients were given at least two drugs simultaneously, and were also put on a diet rich in Vitamin-B. The results were disappointing.

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BRONCHOESOPHAGOLOGY COURSE.

The next Bronchoesophagology Course to be given by the University of Illinois College of Medicine is scheduled for the period, March 22 through April 3, 1954. The course is under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

MALIGNANT MELANOMA OF THE NASAL SEPTUM.

FRED W. ALEXANDER, M.D.,

Cleveland, Ohio.

The purpose of this paper is to report what I believe is the twenty-fifth case of malignant melanoma of the nasal septum. Of all malignant melanomas of the nasal cavity and sinuses, there are only 93 cases mentioned in the English literature. In many of these cases the site of origin is vague, but 24 cases are definitely cited as originating on the nasal septum. Pipia, cited by Grace,[1] states that these tumors never arise in the superior portion of the nasal membranes, but always arise in the lower portion of the nasal septum and middle or inferior turbinate. I find that this is essentially true in the cases reported to the present date, though Pipia made his observation in 1927.

CASE REPORT.

On March 6, 1948, a white, 59-year-old female presented herself with the complaint of being unable to breathe through her nose for four months. Past history revealed that she had seven nasal polypectomies in the last 25 years. In 1940, she had an operation on her forehead for what may have been an osteomyelitis of the frontal bone. Nose bleeds were intermittent in the past, and because of these and/or mild hypertension, she was under mild sedation and on a severe diet that had caused her to lose 20 pounds. A goiter had been present for 20 years, and other than the nasal polypi, the patient stated that she had been in good general health.

Examination revealed a cruciate incision of the forehead in the midline, with a slight depression of the outer table of the forehead. No glands were palpable in the neck. A large, movable, very hard goiter presented itself in the suprasternal notch. The left naris contained many polypi, with complete obstruction of the airway. The right naris was also obstructed with many large nasal polyps, and showed signs of recent bleeding. The polypi in the right naris protruded into the vestibule, bled easily when touched, and were of a normal fish-flesh color. Mouth, tongue and pharynx were normal. There was much mucoid discharge in the nasopharynx, but the posterior naris, Eustachian tube orifices and palate were normal. The patient was edentulous with full upper and lower dentures. The larynx was normal in appearance and function. Large tonsils were present, but not infected. Both ears were normal with intact drums and normal hearing on audiometric examination. Sinuses were clear to transillumination. Chest was clear to auscultation and percussion; heart was normal in size and sounds; blood pressure 180/90. No masses or glands palpable anywhere on body. Chest X-rays normal. Weight 118 pounds.

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Course: On March 8, 1948, the patient was admitted to St. Luke's Hospital, and multiple nasal polyps were removed from both nares and ethmoid sinuses. During the course of the operation a darkly pigmented livid-brown sessile 1 cm. mass was noticed on the right side of the nasal septum located 2½ cm. from the mucocutaneous junction opposite the anterior end of the middle turbinate, 2½ cm. from the floor of the nose, in the mid-septum. This mass was raised from the surface slightly and bled when touched. Because of its unusual appearance the mass was biopsied and the base obliterated with actual cautery. The mass did not seem to extend below the mucosa, and was not connected to the cartilaginous septum. The pathologist reported the biopsy as malignant melanoma, and the other excised material as nasal polyps. On March 12, 1948, under cocaine anesthesia, with a Bovie knife the entire nasal septum was removed *enmasse*, giving a wide 2½ cm. margin of normal tissue between the incision and the tumor edges. This was examined by the pathologist in a very careful manner, and no evidence of tumor could be found on the resected portion of the septum or in the area of cauterization at the site of the tumor. Postoperative recovery was uneventful, and the patient was discharged, but was kept under observation at frequent intervals.

Six months later, September 2, 1948, following an acute upper respiratory infection, the patient developed swelling of the forehead, and was admitted with a diagnosis of acute osteomyelitis of the frontal bone on the right. This was confirmed by X-ray studies, and September 3, a radical removal of both frontal bones and obliteration of the frontal sinuses was performed. Microscopic examinations of the tissues removed revealed osteomyelitis and infection without evidence of any tumor. The nose was clear of tumor, and no masses or suspicious areas could be seen on any of the visible surfaces of the mucosa of the nose, septal perforation, turbinates, or nasopharynx. Recovery was uneventful, and patient was kept under observation at monthly intervals until September, 1949. During the year she maintained a weight of 140 pounds.

Nineteen months after the discovery of the initial lesion of the septum, September 24, 1949, the patient complained of slight bleeding from the throat and mild obstruction of breathing through the left nostril. On examination a small darkly mottled mass about 3 cm. in diameter, which bled easily on palpation, was seen protruding from the left side of the vault of the nasopharynx. This mass was removed under local anesthesia and proved to be malignant melanoma. No glands were palpable in the neck or elsewhere. X-ray survey of the neck, sinuses, skull, and chest were normal except for old operative defects. Two months later the mass in the vault of the nasopharynx again became noticeable but was not touched until February 6, 1950, when the mass had become pedunculated and started to bleed. The mass was then removed by simple local excision under general intratracheal anesthesia, and again proved to be malignant melanoma. The patient's weight remained at 148 pounds, and no evidence of tumor could be seen in the nasopharynx or elsewhere until June 24, 1950, when a small mass appeared in the vault of the nasopharynx over the right eustachian tube. This was watched until September, 1950, when a large node appeared high in the left side of the neck and another node in the right axilla. No therapy was given, but observation continued until February 27, 1951, three years after discovery of the initial lesion, when the mass was again removed from the nasopharynx because of bothersome bleeding. No local recurrences could be seen in the nose or about the nasal septal perforation following this operation. The large node in the left side of the neck and in the right axilla gradually disappeared completely, after being present for 22 months. The patient weighed 155 pounds on June 30, 1951.

In July, 1951, the nasopharyngeal mass recurred to an extent with sufficient bleeding to require removal again for the fourth time, and the nasopharynx remained clean until October 18, 1952, at which time a severe respiratory infection caused the right side of the forehead to swell and the right eye to close, but the infection subsided under antibiotics without further surgery. At that time a small mass was noticed recurring in the right nasopharynx, but did not cause sufficient symptoms or bleeding until further extension over a gradual period of time began to involve the right posterior naris and posterior ethmoid area. This gradually spread into the right ethmoid sinus area until bleeding became a problem. Again, and for the fifth time, the mass was removed from the nasopharynx and right ethmoid area on December 12, 1952, four years after initial discovery. Despite these many vicissitudes, the patient remained well and active physically between bouts, with an average weight of 148 pounds, until January, 1953, when the right anterior ethmoid area began to bleed from a recurrence of the mass in that area. The nasopharynx remained clear of tumor. On March 27, 1953, a large mass was removed from the right ethmoid area under general anesthesia and bleeding was severe, being controlled with difficulty. At this time it was thought that the cribriform area had been involved, but the patient quickly recovered and gained weight up to 150 pounds and went about her business with vigor until June, 1953.

June 9, 1953, five years and four months after the initial lesion's discovery, the patient complained of persistent headache of vague location and developed more bleeding from the right ethmoid area. The tumor from the ethmoid sinus extended up into the cribriform area and upper septum. Bleeding was controlled with intermittent packing for a time, as it was thought we were nearing the end of the road and that further surgery would be futile; however on June 27, 1953, bleeding became so severe that the patient was admitted and the nose packed. On July 1, 1953, bleeding became uncontrollable and patient was taken to surgery and the easily accessible portions of the tumor were removed from the right ethmoid and septal area, where it was obvious that the tumor had penetrated the cribriform area toward the brain. Postnasal packs and heavy vaseline and cellulose packing to both nares controlled the bleeding. To our surprise we were able gradually to remove all packing, and the patient left the hospital by the tenth postoperative day. The patient continued to have headaches, but not too severe to prevent her getting about and doing light work until August, 1953, at which time bleeding gradually became more pronounced as the tumor in the right ethmoid and cribriform area grew more massive. On August 29, 1953, for the eighth time, the patient went reluctantly to the hospital because of severe bleeding, and a huge mass of tumor was removed from the right ethmoid and cribriform area. The cribriform area was markedly involved and its line of demarcation poor. Much necrosis and infection was apparent in the area. The area was packed to control hemorrhage and the patient did well for two days, and packing was removed without bleeding. Three days postoperatively the patient became disorientated and developed a high fever, without stiff neck, and we suspected brain abscess; however, despite supportive treatment and antibiotics, the patient did not rally, and on the tenth postoperative day showed 2000 cells in the spinal fluid with definite signs of meningitis. This progressed rapidly with severe pulmonary edema and death on the twelfth postoperative day, September 10, 1953, five years and six months after discovering the malignant melanoma of the nasal septum.

Autopsy report summary is as follows: Malignant melanoma of the nasal septum with direct extension to the nasopharynx, paranasal sinuses, and the base of the brain. The brain showed a mass 2.8 x 3.1 cm., with diffuse mottling of brownish-black pigment extending into the lower

portions of the frontal lobes 3.5 cm. with a necrotic area involving the center of the tumor. No distant metastases were found in the lymphatic or other systems.

Figure 1 shows a typical section of the malignant melanoma.

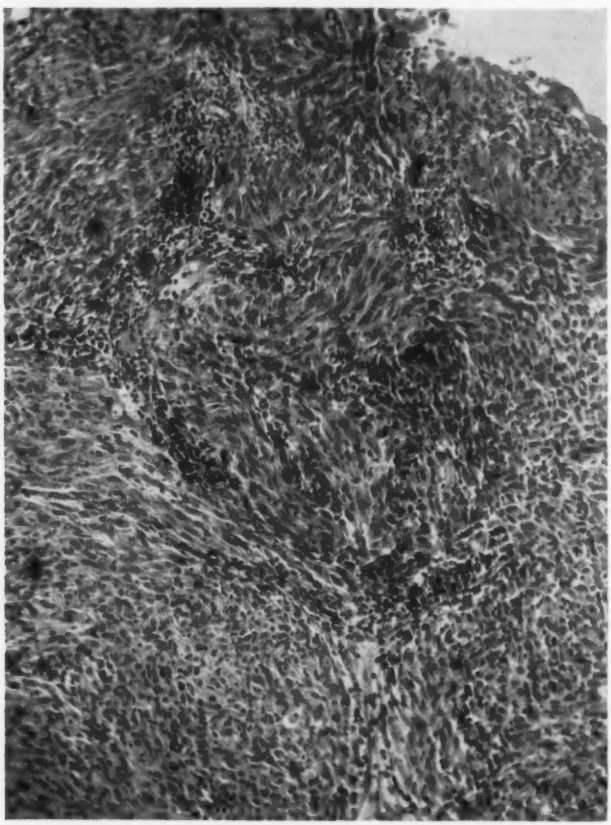

Fig. 1. Typical high-power section of the malignant melanoma, from the nasal septum.

COMMENTS.

Adequate review of the literature and cases reported up to 1947 is given by Grace.[1] Alsup[2] gives the subject good cov-

erage through 1949. These two papers cover all malignant melanomas of the nasal cavity and adjacent sinuses reported in the literature, through 1949, *i.e.* 69 cases. Only 13 cases appeared in American literature until 1949. Since 1949 there are 24 other cases reported in the English literature as follows: Schoolman and Anderson,[3] six cases in negroes; Morris and Horn,[4] one case in a negro; Morey,[5] one white case; Capps and Williams,[6], one case; and T. S. Stewart[7], 15 cases; making a total of 93 cases involving the nasal cavity and sinuses. Only 24 of these cases are definitely located on the nasal septum. Many are poorly located as to exact site, but are referred to as in the nasal cavity or sinuses. In general, the most common sites are the lower anterior half of the septum, anterior halves of inferior and middle turbinates, inferior meatus, and floor of the nose. Involvement of the sinuses is much less common than in carcinoma, which shows up in the middle meatuses. Several melanomas are reported that seem to originate far back in the floor of the nose, visible only with posterior rhinoscopy.

Nearly all cases are associated with nasal polypi, and are discovered during operation for removal of the latter. In a few cases the tumor was discovered by the careful scrutiny of a dark colored or freckled area that bled easily on touch. Bleeding and distention of the naris by an intranasal mass is a very common early symptom. It is obvious from reported cases that many of the tumors are not recognized from the appearance of the polyp, or sessile mass, and that the tumor was not recognized until it rapidly recurred with disastrous results. Hence it follows that in all nasal polypectomies the tissues removed should be subjected to miscroscopic pathological examination. From the numerous recent reports, it is also obvious that malignant melanomas of the nasal cavity are far more common than we suspect. Stewart points out that 4 per cent of 300 malignant tumors of this area were malignant melanomas, 80 per cent carcinomas, and the rest malignant tumors of other types.

The prognosis in these tumors is poor for ultimate survival, but 5 per cent may survive from two to five years. Once the lesion has been operated upon or disturbed, the growth seems to accelerate and may metastasize to any area, or recur only locally at a rapid rate; however, it is a fact that if they survive the first two years, they may go several more years (up to five years) without too much difficulty. Strange as it may seem, the cases that survived the longest were those having frequent local excisions of recurrences. Alsup reports a case that lasted seven years and three months. The prognosis of these tumors originating on mucous membranes is poorer than those occurring in the eye or skin, where long periods may elapse between the appearance of the "latent metastases." In tumors originating on the mucosa of the nasal cavity the periods of quiescence of the latent metastases are short, the longest of the recorded cases being three years.

Treatment by radiant energy (X-rays, radium, etc.) of tumors of this type, and in these areas, is of no value at the present time. This is true of melanomas elsewhere. Packs'[8] principle of "dissection in continuity" is still the procedure of choice in these tumors, and should be practiced despite the difficulty encountered with these cases due to their position in the nasal cavity. This principle should also be followed despite the fact that recorded cases that have had repeated local excisions survive the longest. I believe that radical excision early would increase the extremely poor survival rate, but that once extension has taken place from inadequate removal, no matter how radical, all hope is gone.

SUMMARY.

A case of malignant melanoma of the nasal septum, twenty-fifth in the literature, is reported.

Malignant melanomas are more common in the nasal cavity than is generally recognized.

Radical surgical excision should be practiced at the earliest possible moment if any hope of ultimate survival is to be obtained in these otherwise fatal cases.

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10515 Carnegie Ave.

THE AMERICAN OTORHINOLOGIC SOCIETY FOR PLASTIC SURGERY, Inc.

The regular and scientific meeting of The American Otorhinologic Society for Plastic Surgery, Inc., will be held on Saturday, March 27, 1954, at Hotel Statler, Boston, Mass.

Following is a schedule of the program:

2:00 P.M.—"The Management of Cleft Palate Cases Involving the Hard Palate, So As Not to Interfere with the Growth of the Maxilla."—Dr. Fred Squier Dunn (by invitation). Discussion—Dr. Raymond Rosedale, M.D., M.Sc. (Med.)

3:00 P.M.—"Surgical Management of Complete and Incomplete Bony Atresia of the Posterior Nares."—Dr. Henry Beinfield. Discussion—Dr. Matthew Ersner.

4:00 P.M.—Display of Special Exhibits.

6:30 P.M.—Cocktails and Dinner.

8:00 P.M.—"The Management of Congenital Cleft Lip and Associated Nasal Deformities."—Dr. Douglas Parker (by invitation).

9:00 P.M.—"Round Table Discussion on Otorhinological Problems." Moderator—Dr. Nathan Bolotow.

For further information write Dr. L. J. Feit, 66 Park Ave., New York 16, N. Y.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

February 1, 1954.

Acousticon Models A-17, A-180 and A-185.
Manufacturer: Dictograph Products, Inc., 95-25 149th St., Jamaica 1, New York.

Auditone Models 11 and 15.
Manufacturer: Audio Co. of America, 5305 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67 and 70.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac Model M; Mono-Pac Model "Lyric"; Mono-Pac Model "Rhapsody."
Manufacturer: Beltone Hearing Aid Co., 2900 West 36th St., Chicago 32, Ill.

Cleartone Model 500; Model 700; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Model D-1; Dahlberg Junior Model D-2; Dahlberg Model D-3 Tru-Sonic; Dahlberg Model D-4 Tru-Sonic.
Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Fortiphone Models 19-LR; 20A; 21-C and 22.
Manufacturer: Fortiphone Limited, Fortiphone House, 247 Regent St., London W. 1, England.
Distributor: Anton Heilman, 75 Madison Ave., New York 16, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.
Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.
Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico Model J; Maico Top Secret Model L; Maico Maxitone.

Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis, Minn.

Micronic Model 303; Micronic Model "Mercury"; Micronic Star Model.

Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Classic Model T9; Microtone Model T10; Microtone Model T612.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 106 So. 7th St., Philadelphia 6, Pa.

Normatone Model C and Model D-53.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40 St., Minneapolis, Minn.

Distributor: Normatone Hearing Aid Co., 22 East 7th St., St. Paul (1), Minn.

Otarion Models B-15 and B-30; Otarion Models F-1, and F-3; Otarion Model G-2; Otarion Model G-3; Otarion Model H-1; Custom "5."

Manufacturer: Otarion Hearing Aids, 4757 N. Ravenwood, Chicago 40, Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny-Mite); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Model 62 Starlet; Model 72; Model 82 (Zephyr).

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Distributor: Radioear Corp., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Silvertone Model H-16, J-92; Silvertone Model P-15.

Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.

Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.
Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966; Sonotone Model 977; Sonotone Model 988.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

Televox Model E.
Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 500; Telex Model 952; Telex Model 953; Telex Model 1700.
Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Tonamic Model 50.
Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster; Model Cameo.
Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill.

Unex Midget Model 95; Unex Midget Model 110; Unex Models 200 and 230.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Models J and J-2.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal; Zenith "Regent."
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices have vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TRANSISTOR HEARING AIDS ACCEPTED.

Acousticon Model A-300; 1 transistor, 2 tubes, and 2 batteries.
Manufacturer: Dictograph Products., Inc., 95-25 149th St., Jamaica 35, N. Y.

Audivox, Model 71; 3 transistors, and 2 batteries.
Manufacturer: Audivox, Inc., 123 Worcester St., Boston 18, Mass.

Maico Transist-Ear, Model O; 3 transistors and 1 battery.
Manufacturer: The Maico Company, Inc., 21 N. 3rd St., Minneapolis, 1.

Otarion Model C-15; 1 transistor, 2 tubes, and 2 batteries (A & B).
Manufacturer: Otarion, Inc., 4757 N. Ravenswood Ave., Chicago 40, Ill.

Sonotone Model 1010; 1 transistor, 2 tubes, and 2 batteries (A & B).
Manufacturer: Sonotone Corporation, Elmsford, N. Y.

Telex Model 954; 1 transistor, 2 tubes, and 2 batteries (A & B).
Manufacturer: Telex, Inc., Telex Park, St. Paul, 1.

Zenith Model Royal-T; 3 transistors, and 1 battery. Zenith Model Super Royal-T; 3 transistors, and 1 battery.
Manufacturer: Zenith Radio Corp., 5801 W. Dickens Ave., Chicago 39, Illinois.

SEMI PORTABLE HEARING AIDS.

Ambco Hearing Amplifier (Table Model).
Manufacturer: A. M. Brooks Co., 1222 W. Washington Blvd., Los Angeles 7, Calif.

Aurex (Semi-Portable).
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago 10, Ill.

Precision Table Hearing Aid.
Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

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Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Statler Hotel, Boston, Mass., May 23-24, 1954.

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President: Dr. LeRoy A. Schall, 243 Charles St., Boston, Mass.
President-Elect: Dr. Kenneth M. Day, 121 University Pl., Pittsburgh, Pa.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Meeting: Statler Hotel, Boston, Mass., May 25-27, 1954. (Mornings only.)

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Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3, Tenn.
Meeting: San Francisco, Calif., June 21-25, 1954.

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Meeting: Waldorf-Astoria, New York City, Sept. 19-24, 1954.

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Meeting: Statler Hotel, Boston, Mass., May 17-22, 1954.
Waldorf-Astoria, New York City, Sept., 1954.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

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Secretary: Dr. F. Johnson Putney, 255 So. 17th St., Philadelphia (3) Pa.
Meeting: Statler Hotel, Boston, Mass. (Afternoons) May 25-26, 1954.

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Vice-Chairman: Dr. Irvin Feldman.
Secretary: Dr. Frasier Williams.
Treasurer: Dr. John Louzan.
Meetings are held on the third Tuesday of October, November, March and May, 7:00 P.M.
Place: Army and Navy Club, Washington, D. C.

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Meeting: Waldorf-Astoria, New York City, Sept., 1954.

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Meeting: Waldorf-Astoria, New York City, September, 1954.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Justo M. Alonso, Montevideo.
Executive Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U. S. A.
Meeting: Fourth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
President: Dr. Ricardo Tapia Acuna, Mexico City.
Time and Place: Feb. 28 to Mar. 4, 1954, Mexico City.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

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Meeting: Chicago, Ill., Sept. 22-24, 1954.

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Place: Los Angeles County Medical Association Building, 1925 Wilshire Boulevard, Los Angeles 57, Calif.
Time: 6:00 P.M., first Thursday of each month from September to June inclusive—Ophthalmology Section. 6:00 P.M., fourth Monday of each month from September to June inclusive—Otolaryngology Section.

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Meeting: Joint, with South Carolina Society of Ophthalmology and Otolaryngology, Durham, N. C., Nov. 4-6, 1954.

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Meeting: Joint, with North Carolina Eye, Ear, Nose and Throat Society, Durham, N. C., Nov. 4-6, 1954.

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President: Dr. Leland G. Hunnicutt, 98 N. Madison Ave., Pasadena, Calif.
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Meeting: Honolulu, 1954.

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Time: June 13-15, 1954.

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Meeting: 3rd International Congress of Broncho-Esophagology.
Time and Place: September or October, 1954, Lisbon, Portugal.

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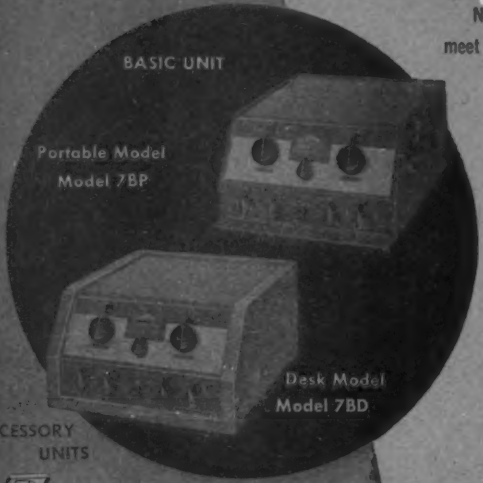

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